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A TEXT-BOOK OF DENTAL PATHOLOGY AND THERAPEUTICS,

INCLUDING

PHARMACOLOGY.

BEING

A TREATISE ON THE PRINCIPLES AND PRACTICE OF DENTAL MEDICINE.

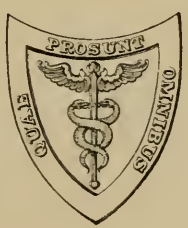
FOR STUDENTS AND PRACTITIONERS.

BY

HENRY H. BURCHARD, M.D., D.D.S.,

SPECIAL LECTURER ON DENTAL PATHOLOGY AND THERAPEUTICS IN THE PHILADELPHIA DENTAL COLLEGE.

ILLUSTRATED WITH 388 ENGRAVINGS AND TWO COLORED PLATES.



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DEDICATED TO

G. V. BLACK, M. D., D. D. S., D. Sc.,

TO WHOM RATIONAL DENTAL PATHOLOGY OWES SO MUCH,

AND TO

DR. EDWARD C. KIRK,

TO WHOM DENTAL SCIENCE OWES MUCH AND THE AUTHOR OWES MORE THAN HE CAN EVER REPAY.

PREFACE.

THIS volume is designed as a text-book of the principles and practice of dental medicine for the use of students, and as a reference work on applied special pathology and therapeutics for the use of dentists. Accepting the dictum of the advanced teachers of the day, the writer believes that an entirely rational system of dental medicine can have but one basis—namely, the same principles which underlie general medical and surgical practice. The book represents, therefore, an attempt at formulating, from data obtained from every available source, a system of dental pathology and therapeutics of which the several parts shall be in harmony with one another and also with the several collateral sciences involved. The impulse prompting the work was no desire to multiply books, but arose from a conviction expressed by many teachers, that such a volume is needed by students, practitioners, and teachers.

The extent and scope of references may be only partially seen in the numerous foot-note references, space limitations precluding any exhaustive bibliography.

It would be unjust, however, to omit this opportunity to credit two investigators without whose researches this volume would have been impossible: Professors G. V. Black and W. D. Miller, to whom frequent and specific references are made.

The immense development of modern dentistry has brought with it a more rational and convenient grouping of its subjects. The *American Text-Books of Operative* and *Prosthetic Dentistry* have already won acceptance, each in its own field. They leave untouched a range of subjects which are naturally cognate, and hence are most advantageously taught in conjunction—namely, Dental Pathology, Therapeutics, and Pharmacology. The fitness of this grouping is manifest.

Special thanks are due the publishing department of the S. S. White Dental Manufacturing Co. for their liberality in furnishing illustrations;

thanks for cuts are also tendered G. V. Black, J. S. Marshall, J. L. Williams, Wm. Wood & Co., and P. Blakiston, Son & Co.

The author tenders to Lea Brothers & Co. his grateful thanks for the more than liberal spirit exhibited by them in the making of the book; from beginning to end their course has been one of great courtesy and unbounded generosity.

H. H. B.

April, 1898.

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DENTAL PATHOLOGY, THERAPEUTICS, AND PHARMACOLOGY.

SECTION I.

GENERAL PATHOLOGY.

CHAPTER I.

GENERAL PRINCIPLES.

The study of dental pathology and therapeutics embraces a consideration of such principles of general pathology and therapeutics as find application in the practice of dentistry, and in addition implies and requires an exhaustive investigation into the modifications of these general principles growing out of the peculiarities of anatomical structure and the functions of the parts included in the field of operation. There is, perhaps, no aspect of surgery in which these modifications of treatment, due to anatomical peculiarities, are more marked than in dental therapeutics.

A consideration of the special practice must, however, be preceded by a study of the general disease-processes which affect the tissues of the body; so that a special application of the knowledge thus gained may be made to render clear the nature of dental diseases and the rationale of their treatment.

The word pathology is derived from the Greek *pathos*, disease, and *logos*, a treatise or discourse. Applied in its general sense, it includes a study of the natural history of diseases, their causes, progress, phenomena, and terminations. The word therapeutics is derived from the Greek *therapeuein*, to take care of, meaning the measures adopted to remedy or remove the changes induced by pathological processes.

Pathology, as has often been stated, cannot be pursued as an independent study, for it is but an expression of other collateral sciences. It is a perversion of the processes found associated with what are termed living bodies; in its full and philosophical sense it is morbid biology—an altered physiology. It follows, therefore, as a corollary, that an exhaustive familiarity with the contemporary state of the science

of physiology is necessary to a clear comprehension of the nature of pathological processes. The more minutely an investigator proposes to examine into pathological states, the more extensive must be his parallel knowledge of physiology. While many phases and features of pathology may be grasped by a student of but two courses, there are problems in modern pathology that test and exceed the resources of the veteran of the physiological, chemical, and pathological laboratory.

Whatever field of medicine or surgery may be under systematic exposition, its study is made more clear by turning to and determining the nature of its logical basis, and proceeding from that as a fixed point. While it is true that pathology is by no means an exact science, for the reason that its mother science "biology" has not yet achieved that position, it is indubitable that year by year it increases in the measure of its exactitude. It is relatively a speculative science when compared with such a science as chemistry, which begins with an approximately fixed unit, the atom, and has, therefore, a corresponding stability of superstructure. A true, exact science, as mathematics, begins with a fixed and unchangeable unit, and is therefore absolute.

The unit of the physiologist, and consequently of the pathologist, is a complexity in itself; it is a substance called protoplasm, of which our knowledge is imperfect and relative; it follows, therefore, that sciences based upon such a unit will be susceptible to changes and alterations until the knowledge of the accepted unit is as complete as the knowledge the mathematician has of his unit.

A contemporary philosopher has said that the standing disgrace of the science of chemistry is that it has not yet given an analysis of protoplasm. Protoplasm, so far as present knowledge permits a statement, is a viscid substance, which under certain conditions exhibits a sequence of phenomena which the physiologist calls life. Its chemical composition is very imperfectly known; indeed, the only means available for its chemical analysis are such as destroy the identity of the substance. More than this, a subtle change occurs with the cessation of vital activity; so that in all probability an analysis of dead protoplasm would not represent the composition of vital protoplasm. As set forth by O. Hertwig,[1] "we must regard protoplasm as a morphological, not a chemical conception;" "it is not a single chemical substance, complex in composition, but is composed of a large number of different chemical substances which we have to picture to ourselves as most minute particles united together to form a wonderfully complex structure." "Protoplasm cannot be placed under different conditions without ceasing to be protoplasm, for its essential properties in which life manifests itself depend upon a fixed organiza-

[1] *The Cell*, 1895.

tion."[1] Huxley has well termed the substance "the physical basis of life." The nearest approach to a chemical conception of the substance is that it is a collection of proteids; and of these substances our knowledge is very imperfect. They consist of carbon, hydrogen, oxygen, and nitrogen, combined with sulphur and phosphorus in enormous molecules. It is known certainly, however, that protoplasm does not behave as a fixed chemical substance.

As studied by the physiologist and pathologist, observations as to its properties are made in defined masses of protoplasm, which exhibit the sum of phenomena called life. A simple mass of protoplasm is studied to determine, as far as possible, the ultimate phenomena relating to vitality. For this purpose an amœba, the anatomical equivalent of a white blood-corpuscle, is placed under conditions which permit of close and continued observation and where the condition of its environment may be altered to study the effects of such alterations upon the vital processes of the cell. The study of collections of cells is a secondary pursuit. While, as has been stated, protoplasm, or contemporary knowledge of it, is the basis of the sciences of biology and pathology, the actual unit of the sciences is the cell. All reasoning proceeds from this basis, that the ultimate phenomena of life are expressed in cell-life, so that the properties and functions found associated with cells are the underlying data of pathology.

If a drop of water be taken from the sides or bottom of an aquarium, placed on a slide and covered with a cover-glass, and then placed under a microscope with, first, a $\frac{1}{4}''$ objective, there will be noted at some portion of the fluid a small transparent mass having the appearance of a colorless fragment of jelly; this is an amœba. The outline of the mass may have almost any form. At some portion there will be a defined and easily distinguished spot, the nucleus; at another point a vesicle is seen; the body of the amœba appears to contain numbers of fine granules. The nucleus appears more markedly granular than the body of the amœba. If kept under observation, the amœba will be seen to change its outlines; at one or more, and it may be in several places projections like blunt arms or feet are seen to be extending from the amœba (see Fig. 1). On account of their appearance they are called by the physiologist pseudopodia, from *pseudo*, false, and *pous*, a foot—false feet. These changes of form are much varied (Fig. 2). The cell has, therefore, the property of altering its form—*i. e.*, it has contractility. If the temperature of the slide be raised, the movements

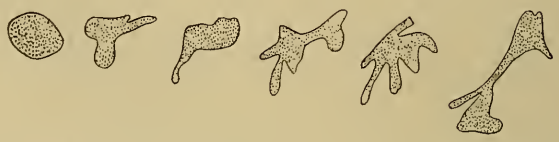
FIG. 1.

Changes of form in an amœba: *P,P*, pseudopodia; *V*, vesicle: *N*, nucleus.

[1] *The Cell*, 1895.

of the cell become more rapid, and if raised to a temperature of 55° C. the cell contracts in a round lump; it responds to stimuli, and has therefore the property of irritability.

FIG. 2.



Amœboid movement of a white blood-corpuscle of man; various phases of movement. (Klein.)

When certain solid substances contained in the water come in contact with the amœba, the latter is seen to flow around and engulf them; as is shown in Figs. 3 and 4, where the analogue of an amœba, a

FIG. 3.

Leucocyte of a frog from the neighborhood of a piece of the lung of a mouse infected with anthrax, about forty-two hours after the piece of lung had been placed under the skin of the frog's back. The leucocyte is in the act of eating up an anthrax bacillus. (Brunton, after Metchnikoff.)

FIG. 4.

The same leucocyte a few minutes later, after it has completely enveloped the bacillus. (Brunton, after Metchnikoff.)

leucocyte, has taken in bacteria. After a time the ingested body is found to have disappeared; it has been digested. Evidently the cell must produce a substance capable of dissolving some foreign substances—*i. e.*,

FIG. 5.

Forms assumed by a nucleus in dividing: *a*, resting nucleus; *b*, skein-form, open stage; *c*, wreath-form; *d*, aster, or star-form; *e*, equatorial stage of division; *f*, separation more advanced; *g* and *h*, star and wreath forms of daughter-nuclei. (Reduced from Flemming's drawings in the *Arch. f. Mik. Anat.*)

it has the function of secretion. More than this, the amœba does not take in substances indiscriminately; some it rejects.

If the observations are continued, it will be noticed that changes occur in the nucleus of the cell. A series of alterations in its figure are noted, as shown in Fig. 5. Two nuclei are formed, and soon the body of the cell divides and two cells appear—the amœba has reproduced itself. This primitive cell has, therefore, the properties of irritability, contractility, secretion and excretion (as will be seen in later studies), and reproduction; moreover, it responds to stimuli, as seen on warming the stage.

If the stage be cooled, the movements of the protoplasm are lessened, and when foreign substances come in contact with the cell it fails to encompass them—its irritability and contractility are lessened.

It is noted that some simple cells are attracted and stimulated by light; others are repelled by it.

If a mild induction (interrupted) current be passed through the water in which the amœba is,[1] the movements of the cell are checked; if a strong current be passed, the cell contracts sharply. If a galvanic (constant) current be passed, movement at first ceases, but pseudopodia are extruded toward the cathode and the cell crawls toward that pole.

The cell responds to mechanical stimuli, such as violent shaking, by contraction.[2]

If substances such as ether or chloroform are added to the fluid, the irritability of the cell is so lessened that it does not respond to stimuli.

If the supply of oxygen be cut off, or if carbon dioxid be admitted to the fluid, movement ceases and the cell remains contracted.

These examples serve to illustrate that protoplasm responds to stimuli of physical and chemical nature, and that its functions may be altered by substances which are brought in contact with it. Upon these facts depends the practice of therapeutics.

A living organism, it will be seen, has a certain degree of action and function. The general average of its action and function is spoken of as a condition of health. When, from any cause, the functions are raised or lowered from the general average, a condition of disease exists.

Stimulation.—Certain agencies applied to the cell increase its activity; this is called stimulation. The movements of the cell become more rapid, food-particles are taken in more rapidly and disappear more quickly; irritability, contractility, and secretion are increased. The cell subdivides, or reproduces more quickly. Increase the stimulation, and the vital activity becomes fretful; in some cases cell-division is incomplete—the nucleus divides, but not the cell-body. Increase the stimulation beyond this degree, and the wearied cell ceases its movements—refuses to respond; is paralyzed by overwork.

Sedation.—If the conditions be reversed; if, instead of applying a

[1] O. Hertwig, *The Cell.* [2] *Ibid.*

stimulus to the cell, an opposite influence be introduced, the phenomena are reversed.

If the temperature be reduced, the movements of the cell become sluggish; the body changes its form more slowly and less extensively—*i. e.*, contractility is lessened; particles taken into the cell remain apparently unchanged; irritability, secretion, and excretion are lessened; and, furthermore, reproduction does not occur nearly so rapidly—that is, the cell in contact with sedative influences has all of its activities lessened.

There are two great classes of influences, then, which affect the vitality of cells: stimulation, which, if continued long enough, leads to death through overwork; and sedation, which, if continued, paralyzes all of the energies of the cell—it is starved to death.

Every cell has a range of resistance to these influences which tend to destroy it, which is fitly termed the resistance of vitality. Disease itself is some alteration in any one or more of these several cell-properties, of irritability, contractility, growth, secretion, maintenance, or reproduction. If any one of these properties is not exhibited, it is said that the cell is diseased.

If a brood of cells derived from one parent be examined, some will be seen to grow more rapidly than others, their movements are more rapid and they reproduce more quickly; others have sluggish functions and movements.

The cell lives its cycle and reproduces, and the parent is no more, the life being continued in the offspring.

The life and properties of this small mass of protoplasm represent in miniature the primitive functions and life of the highest animals.

The contractility is represented in the motive apparatus of the higher animals.

The reception, engulfing, and dissolving, or casting out, bodies with which the amœba is brought in contact correspond in the higher animals with the digestive apparatus and process and the excretory function.

The highly evolved irritability is represented in the nervous system of the higher animals.

The movements occurring in and about the vacuole are the progenitors of the circulatory apparatus and all of its adjunct organs.

If the irritability of a simple cell be increased or diminished, it corresponds with a disease of the nervous system, and so with the other functions.

It is essential to the life of a cell that the means be afforded it of continuing the activities called life. First, it must receive a proper food-supply, which includes an abundance of water and such solid substances, either contained in the water or dissolved in it, as are necessary to replace loss of substance in the cell; these are albuminous substances,

to replace used-up nitrogenous matters; carbohydrates and hydrocarbons, to replace the everchanging proportions of carbon and hydrogen of living matter; and, lastly, the supply of oxygen must be sufficient. The cell must be maintained at a proper temperature. Its waste-products must be removed. If any of these conditions be interfered with, vitality is depressed. The most certain cause of prompt death is an absence of oxygen. Cell-life is maintained largely by a process of oxidation.

In the performance of any vital act chemical change is inevitable; that is, some portion or amount of protoplasm undergoes chemical alteration; the substances brought as food by the circulating fluid are built into cell-substance to replace those which are lost; the setting free of chemical energy by the process of protoplasm breaking down into simpler bodies is manifested in the production of heat.

The oxygen about the cell combines with the products of cell-activity, and changes them into substances from which the cell frees itself—the waste, the products of its activity, is removed. The products of cell-activity are largely acid in reaction, and the increase of acid in protoplasm lessens its affinity for oxygen; hence in an overworked cell oxidation is sluggish, for the outer portions of the cell have a lessened alkalinity.

It is important that a familiarity with cell-properties be obtained, because the functions of any body are made up of the functions of its cells. As before stated, in the more highly evolved animals special collections of cells and special areas are set aside to perform the functions in which the entire substance of an amœba participates.

Certain collections of cells are found in which one function is active, the others in abeyance; thus, large colonies of cells exist in which contractility is the dominant property noted; these are muscle-cells. Others have but the property of irritability; these are nerve-cells. Still others develop peculiar chemical functions, and become glandular or secretory cells. Such collections of cells are known as tissues. These special cell-colonies or tissues are built together into defined masses for the performance of their specialized functions. In the development of these masses, means of holding and maintaining the cells in definite mass-forms and provisions for their food-supply and waste-removing apparatus are provided in what are called the connective tissues, binding the cells in definite forms and transmitting their vascular supply (food- and waste-carrier). When thus bound together the tissues are said to form organs.

In studying disorders which may affect any one or more organs, or their parts, the problem becomes apparently complex, but it ultimately resolves itself into a study of the disorders of the cells of the part. A disease may only be evident through disorder of some function, for

when the tissue affected is studied no evidence of disease is seen even under the highest powers of the microscope.

Another class of diseases will exhibit changes which have occurred in the anatomy of the part, which prompts the classification of diseases into functional and structural. Functional diseases are those which exhibit no demonstrable alteration in the anatomy of a part. Structural diseases are those in which the anatomical structure of a part is altered.

Definite anatomical changes have been found constantly associated with a more or less constant group of symptoms. The changes are spoken of as the morbid anatomy of a disease.

The pathology of any disease means the morbid physiology through which these anatomical changes are brought about. In the absence of direct anatomical examination of any organ a knowledge that it is not performing its work properly and that its functions are disordered is obtained by noting whatever phenomena are obtainable. These are, in part, what the observer may note through the use of his senses, and by utilizing the resources of physics and chemistry. Knowledge gained by such means is called the objective evidence of disease. Certain facts are elicited by questioning a patient as to altered sensations, the nature and situations of pain, etc.; these are known as the subjective evidences of disease.

The study of symptoms elicited from the patient by examination and noted in his physical appearance is known as Semeiology, from *semeion*, a mark or sign.

A special study is made of the influences and conditions through which the functions of the body are disordered; this is known as Etiology.

It is noted that diseases presenting a certain or an uncertain morbid anatomy and a constant course of symptoms have from their beginning to ending tolerably constant phenomena; they have each a natural history; this is called the Clinical History of a Disease.

There are symptoms which are common to many disease-conditions, and others which are associated with but some one distinctive morbid state; different diseases may exhibit several symptoms in common, and some few, which serve to distinguish the one from the other. The study which deals with that discrimination between groups of symptoms which characterize distinct diseases is called Diagnosis, from Greek *dia*, through, and *gignosko*, I know. It embraces a consideration of all of those phenomena which indicate the exact disease-state present.

In the study of the clinical history of diseases it is observed that while each disease has a more or less well-defined natural history, they vary as to the course followed in individuals and the final outcome. It

is observed that there are signs and symptoms which are followed by good or ill results, as the case may be. By these signs and symptoms it may be foretold with some degree of assurance what will be the probable outcome of the disease. The branch of inquiry dealing with this aspect of disease-study is known as Prognosis, from *pro*, before, and *gignosko*.

It is found that agencies described in the succeeding chapter as disease-causes affect tissues and organs in such manner that their physiology is altered and changes occur in their anatomical structure. The occurrence of these disordered functions and structures is attended by signs and symptoms which indicate their presence. The study of these particular features of disease is called Pathogenesis; while it might be inferred from the etymology of this word (*pathos*, disease, and *gennao*, I produce) that it was but another name for etiology, it covers a wider field.

Recognizing by the presence of signs and symptoms that the physiology of one or more organs is disordered, and that probably changes of structure have occurred, the great problem of the healing art is to apply such measures and agencies as shall restore the tissues or organs to their normal condition and function. This branch of medicine is known as Therapeutics, from *therapeuein*, to take care of, to heal.

Centuries of observation have shown that there are numerous agencies and substances which are capable of producing alterations in the action or physiology of organs; these agencies and substances are included in what is called the Materia Medica—the materials of medicine.

An increasing importance attaches to that method of studying the feature of the healing art which begins with a knowledge of the exact chemical composition of each substance used, and traces with the utmost care the effects of these substances upon the chemical composition and the functions of organs, and how these effects may be utilized in combating disease; this study is called Pharmacology.

The method of treating disease which begins with an exact knowledge of the causes of diseases, the significance of their symptoms, and their clinical history, together with the nature of the alterations of the physiology of organs which give rise to symptoms, and the reasons and nature of the anatomical changes which result from this altered physiology, and embracing an exact knowledge of the effects of agents in the materia medica, which properties are to be utilized in such manner that the causes, phenomena, and results of disease are neutralized, is called Rational Therapeutics.

Such precise familiarity with the nature of disease, and, upon the other hand, of the action of therapeutic agents, is not a possession of

medicine; although it had been noted and confirmed by years of observation that the application or administration of certain agents is followed by a disappearance of symptoms and of the evidences of disease; the type of treatment pursued upon this basis is called Empirical Therapeutics.

When it is observed that patients exhibit groups of symptoms which appear as constant features of clinical histories, which are alike, and it is further noted that the causes giving rise to these symptoms are identical, and when, furthermore, examination shows the same organs to be affected in one manner, a distinctive name is applied to describe the condition; it is known, or designated, as some distinct disease. For example, a number of persons who drink water from a common source are, one by one, affected by diarrhœa and a gradually rising temperature of the body, followed by evidences of debility and general poisoning, which symptoms subside after about three weeks: the patients are said to have the distinctive disease, typhoid fever.

It is preferable, when it can be done, to name a disease to accord with its anatomical and physiological features. For example, chemical examination of a patient's urine shows it to contain albumin; examined under a microscope it shows what are called tube-casts; at a later period, dropsy or œdemas (which see) appear, and the patient exhibits evidences of poisoning, and later dies. On examining the tissues of the body it is found that the secretory or parenchymatous portions of the kidney are destroyed; the disease is designated then as parenchymatous nephritis. This word is derived from *parenchuma*, the substance of organs, and *nephros*, a kidney, the termination *itis* signifying inflammation. This suffix always means inflammation; for example, inflammation of a joint is known as arthritis, from *arthron*, a joint; pulpitis, inflammation of a tooth-pulp, and so on.

Some disorders can only be named from a symptom, as neuralgia, from *neuron*, a nerve, and *algos*, pain. The suffix *algos* always signifies pain in the organ or tissue named in the stem of the word, as odontalgia, stem *odont*, from the Greek *odous*, a tooth.

Pains of a rheumatic character are described by the suffix *dynia*, from *odune*, pain, as in pleurodynia, from *pleura*, the side, and *odune*, pain.

Pains of gouty origin are named by the suffix *agra*, a seizure, as podagra, from *pous*, *poda*, a foot, and *agra*, a seizure, which describes gout of the foot. Odontagra would be the name applied to gouty pain in or about a tooth.

Before the diseases or disorders of any tissue or organ can be studied intelligently it is necessary that the student be familiar with the anatomy of the part, as revealed by the scalpel, or macroscopic

anatomy, and as revealed by the microscope, the histology of the organ and the tissues of which it is composed, or its microscopic anatomy. It is necessary that a knowledge of its physiology be acquired; the nature of the work performed, how and by what performed. The nature of the chemical changes which occur in tissues and organs should also be known; and, finally, the mode of development, the embryology of the tissues and organs, should be studied.

It is customary—in fact, almost necessary—that a disease be studied systematically in all of its bearings. First, What is the definition of the disease? Secondly, How has it been studied in the past?—*i. e.*, What is its history? A study of etiology, or the causes of the disease, follows. Next, What is its semeiology?—*i. e.*, What are its symptoms? Next an inquiry is directed to the nature of the alterations in the physiology of the parts involved, and what changes of structure occur—*i. e.*, a study of the pathology and morbid anatomy of the disease. The clinical history is next taken up, and followed by a consideration of its diagnosis; its prognosis or probable outcome is next adjudged; and, finally, the questions of How shall the disease be combated? What shall be its treatment?

With an increasing knowledge of the pathogenesis of diseases, it is but natural that it should lead to the study of the rational methods of preventing disease—*i. e.*, their prophylaxis (Greek *pro*, before; *phulasso*, I preserve; and *phulax*, a guard). It is manifestly preferable to prevent disease than to permit its onset and treat the condition. Before intelligent efforts can be directed toward the prevention of disease a knowledge of disease-causes is necessary, for the prophylaxis of any disease is the destruction or neutralizing of its causes before they have opportunity to act.

CHAPTER II.

CAUSES OF DISEASE, GENERAL AND LOCAL.

A DISEASE-CAUSE may be defined as any influence of whatsoever nature which is capable of disturbing the nutritive balance of any portion of the body. That branch of study which deals with the causes of disease is called Etiology, from Greek *aetios*, causes, and *logos*.

Every living organism and all parts of the more highly organized animals possess the power of maintaining a nutritive balance despite changes in their surroundings; moreover, they persist as living bodies though assailed by forces which tend to disturb the complexus of phenomena called life, even though these forces do frequently bring about modifications in their capacity for work. All of these threatening influences may be regarded as disease-causes, and an examination of the forces which tend to disturb the natural working of portions of the body will show them to be very numerous; but, as will be seen later, they may be grouped under a few convenient headings.

This study deals with the forces and influences which act upon the body from the outside; they are called the extrinsic causes of disease.

The evolutionist points out that all animals of a species are not equally resistant to the same causes acting upon all, for if a number of animals be placed under identical conditions, some will be more strongly affected than others; some are, therefore, said to have a greater inherent resistance than others. The reason for this difference in resistive power is spoken of as intrinsic; the causes of the lessened resistance, as the intrinsic causes of disease. The cause of the condition may be sought for in all three of the factors which go to make up the analysis of cell-life, chemical, physiological, and anatomical; although it is at times difficult or even impossible to determine exactly which element is at fault. An examination of the part may reveal some structural change, some alteration of its anatomical forms; or, again, it can only be said that the part does not functionate properly, or it may be that the evidence of disorder is a persistent deficiency in the chemical nature of cell-products. It is impossible to separate the physiological from the chemical side of this problem.

The only present, tangible conditions which can be regarded as purely intrinsic disease-causes are changes in anatomical structure. Changes or conditions which alter the physiological action of any part of the

body, so that it succumbs to influences which normally it should resist, are called predisposing causes of disease. It is evident that what are termed intrinsic causes are all predispositions. It is customary, therefore, to divide the study of disease-causes under two heads: first, all of those conditions which render any part more susceptible to the action of inimical forces, or the predisposing causes of disease; secondly, those to whose action upon the body disease is directly due, or the exciting causes of disease. A predisposing cause may be intrinsic or extrinsic, which is also true of the exciting causes, so that we may divide both the predisposing and the exciting causes into intrinsic and extrinsic.

Predisposing Causes of Disease.

Life in itself is a persistence of energy in a chemical substance of unknown nature, against forces which tend to disturb its chemical integrity, and, as a consequence, its energy or its structure. Any forces or influences acting from within or without an organized mass of the substance, which lessen its resistance to those forces, predispose to its destruction. Applied on a large scale, there comes the study of the influences which tend to lessen the resistive forces of the body. The first of these is naturally an alteration of anatomical structure, for if there is a standard of structure best adapted to meeting and overcoming conditions to which living bodies are subjected, any variation in this standard is necessarily followed by lessened resistance.

These alterations of structure may be congenital or acquired; they may be due to a faulty development which may be traced to the ovum or the spermatozoa of the parent, or they may arise after birth and a period of apparently normal development. The abnormalities may be macroscopic, so evident that they are seen at a glance to be malformations; or they may be microscopic, requiring special preparation of portions of organs to demonstrate their existence.

Sex as an Intrinsic Predisposing Cause.—In this connection the influence of sex upon predisposition to disease must be considered. While the general resistive power of the bodies of both sexes may be regarded as practically equal under similar conditions, yet the anatomical structures and physiology of each sex have an influence upon predisposition to certain diseases. Aside from the diseases peculiar to sex, on account of their peculiar organs, each sex exhibits predispositions to diseases which the other sex escapes; for many of these the habits of life furnish an explanation, for others an explanation is not available. For example, while women are predisposed to functional and emotional disturbances of the nervous system, such as hysteria, they are almost exempt from such structural nervous diseases as locomotor ataxia and general sclerosis.

Men may become anæmic from a variety of causes; but young girls become chlorotic, a disease-type which rarely affects the male.

Women are affected by diseases of internal secretory glands, as the thyroid, and escape affections of others, as of the suprarenal capsules.

Age as an Intrinsic Predisposing Cause.—The cycle of life of an organized being comprises an intra- and an extra-uterine development until maturity is attained; while increment exceeds excrement, growth is in progress. This is followed by a period in which increment and excrement are balanced; waste and repair are equal; this is the period of maintenance. Succeeding is the period of decadence, when the tissues of the body undergo gradual changes leading to exhaustion; the vital forces are lessened; the power of repair does not equal the waste occurring throughout the body. In each of these periods there is exhibited a predisposition to certain diseases.

Intra-uterine causes acting produce abnormalities of structure—a general intrinsic predisposition. During the first period of extra-uterine growth there is a predisposition to gastro-intestinal disorders and reflex disturbances of the nervous system. At a later period there is a predisposition exhibited to the eruptive fevers, scarlatina, rubeola, varicella, and variola, and also to diphtheria—diseases which are called the diseases of childhood. Later, in the period of adolescence, other predispositions occur in young girls, notably chlorosis. Later come the diseases of early maturity, such as typhoid fever. Diseases characterized by degenerative changes in the tissues and organs are so distinctive accompaniments of senility that they are called the diseases of old age.

Temperament as an Intrinsic Predisposing Cause.—The influence of temperament upon disease is frequently overlooked. Accepting as a basis the classification of Hippocrates as indicating the four classes of temperament, it may be said that persons of the sanguine temperament have a predisposition to acute pulmonary and cardiac diseases, together with those of the bloodvessels; inflammatory disorders are disposed to run a riotous course. Those of the nervous temperament, to disorders of the corresponding anatomical system, particularly functional disorders. Those of the bilious temperament exhibit a predisposition to affections of the liver. Those of the lymphatic temperament have a predisposition to passive congestions. The truth of these rules may be more apparent than real, for close scientific studies relative to the subject of temperament are extremely meagre.

Heredity.—Certain diseases exhibit a predisposition to descend from parent to child; as, for example, gout, rheumatism, syphilis, the tubercular diathesis, epilepsy, etc. The mode of transmission is in all probability the inheritance of a type of tissue; a tissue anatomy and phys-

iology which permit the more ready action of the exciting causes of these maladies. These cases might perhaps be fitly included under the head of malformations, though the malformations may be undiscoverable. Persons who exhibit this tendency to an hereditary disease such as gout are said to have that diathesis. The hereditary victims of such diseases as syphilis are said to be cachectic.

Existing Disease.—The presence of one disease may predispose to others, as, for example, acute rheumatism predisposes to organic disease of the heart-valves; however, in this as in many diseases, what appear to be supplementary diseases may be but phases of the same disease.

Previous Disease.—Frequently a part which has once been the seat of disease is, when apparently recovered, the seat of the same disease or other disease at a subsequent period. Previous disease may, on the contrary, confer immunity from the same disease in the future. This principle is of wide and increasing application in medicine. Notable examples of this are found in smallpox (variola) and in scarlet fever (scarlatina), and to a less extent in typhoid fever.

Extrinsic Predisposing Causes of Disease.—Under this head are included all of those conditions which cause a person to fall a victim to a disease when exposed to its active causes. Cold and damp are among the conditions which act as predisposing causes of disease, as, for example, to pneumonia, rheumatism, and a number of inflammatory disorders. Dentists note how cold and damp act as predisposing influences in the induction of pericementitis. The reasons for this will appear in the discussion of hyperæmia.

Immunity.—When a number of persons are exposed to the active causes of disease it will be seen that some succumb to the disease immediately; others are unaffected—*i. e.*, they are immune. Some are not affected no matter how prolonged the exposure; others exhibit a tardy response to the action of the disease-cause. The reasons for these degrees of natural insusceptibility are but imperfectly understood.

Immunity from a particular disease may be inherited or acquired. It may be exhibited toward only one disease. In some, disease appear to be influenced by sex—as, for example, males have a general immunity from goitre; females, immunity from Addison's disease. Immunity is influenced by age in certain diseases; for example, the diseases of childhood are rare in the elderly; they do occur, however. The degenerative diseases of old age are almost unknown in childhood. Race has its influence; the negro is peculiarly susceptible to smallpox, and almost immune from malaria.

Natural immunity is a very uncertain quantity; there is no method of determining its duration without direct exposure.

The most interesting and useful feature of immunity is the possibility of its acquirement through natural, or its induction through artificial means. It had been noted, as long ago as certain conditions were recognized as definite diseases, that those who recovered from them were rarely the subjects of the same disease in the future, although exposed to actively contagious influences. The classical example of this acquired immunity is smallpox. We are indebted to Saracen medicine, adopted or followed by the Turks of Constantinople, for the early studies as to artificial immunity from this disease. Noting, no doubt, that virulent contagion was associated with the pustular stage of smallpox; that immunity of some persons did exist; and that the disease was self-protective: a minute portion of the virus was introduced into the bodies of healthy persons. This induced in most of them the disease in a milder form, and, although the death-rate was considerable, an acquired immunity was the result. In 1718 Lady Mary Wortley Montague introduced the practice into England, where it met with much opposition and limited adoption. It may be remarked that the mortality of the cases inoculated is but from 3 to 5 per 1000. In 1798, Jenner, an English physician, applied the rustic knowledge that persons who had been inoculated with the virus of cowpox were insusceptible to smallpox, and became the virtual founder of inoculation by attenuated virus. This practice in its later phases is one of the most important developments of modern medicine.

The exact method through which inoculation brings about immunity can at present only be surmised. (See Bacteria (alexins).) It is important to remember, as illustrated in the principle of vaccination, that living bodies have protective mechanisms against disease which undergo variations in degree of activity, and that this activity is in some cases susceptible of artificial alteration.

Exciting Causes of Disease.

Under the head of the exciting causes of disease are included all of those variations in bodily and cellular environment which interfere with the normal performance of function. The conditions of cellular life are, that cells must receive a proper food-supply, must have removed the waste-products resulting from their life-processes, and must be maintained at a proper temperature. In a complex organ the process of nutrition is more complicated, and a greater number of elements must be studied, for not only are the food-supply and waste-removal relegated to special structures, but a nervous element is introduced.

All of those causes which relate to food-supply, all of the processes which food undergoes while being changed into substances fit for cell-nutrition, the removal of waste, and also the supply of oxygen, may be

grouped under the head of abnormal blood-supply. In studying the influences which are grouped under this head, every stage through which the food passes in being changed from albumins, fats, and starches, into material to be utilized by cells, the composition of the blood, its mode of distribution, its supply of oxygen, the removal of waste-substance from cells and its elimination from the body, must be taken into consideration.

Abnormal Food-supply.—When a man has attained maturity waste equals repair in the body; sufficient food is taken into the body, carried to the organs and utilized by their cells to replace the substance lost through functional activity, the waste-substances being oxidized by the oxygen carried by the red blood-corpuscles; carbon dioxid, urea, and other waste-substances are formed, which are immediately removed from about the cells. Each cell is a chemical laboratory in miniature, and any variation in the nature of the substances present produces changes in the chemical interchanges or reactions.

It is evident, therefore, that the study of abnormal food-supply is an extensive one, for the cause of the presence of improper pabulum may be traced to any stage of nutrition from the non-reception of the proper amount and quality of food and its imperfect mastication, to faults connected with glandular secretion of the alimentary tract, to faulty operations while the digested and absorbed food-stuffs are in the portal system; next, to the general character and condition of the circulatory system, to the time when the pabulum is in the lymph-spaces. Any abnormality of function in any one of these particulars may be followed by imperfect nutrition of cells.

Waste-products as Disease-causes.—For the normal performance of cell-function it is quite as essential that cells should have their waste-products removed as it is that they should receive a sufficient supply of pabulum and oxygen.

Waste-products of cell-action are the ashes of the vital fire; are oxidized products of cell-contents which have a simpler chemical composition than the pabulum or cell-materials out of which they are formed. Principal among these products are carbon dioxid and the product of albuminous waste—urea. Numerous other substances are formed, such as xanthin, uric acid, etc. If when these substances are formed, they are not removed, they act as ashes in the vital furnace, clog the fuel, and lessen oxidation. Retained, they inevitably interfere with the nutrition and function of cells, and disease results. If taken into the blood and through faulty action of the great eliminant organs—the kidneys and lungs—they are not removed, they act as disturbing elements throughout the body; for example, should the functions of the kidney be destroyed through disease, urea is not eliminated, and, remaining in the

circulating fluids, causes symptoms of poisoning—uræmia. Uric acid is normally formed in small amounts; if formed in increased amount and not eliminated, owing to disease of the kidneys, it gives rise to a long chain of symptoms, characteristic among which are those comprised under the head of the several forms of gout. It is extremely probable that many diseases of obscure nature may be due to the retention and circulation of waste-products.

The Presence of Poisons.—The presence of introduced poisons in the body, such as mineral and vegetable poisons, must be regarded as disease-causes: for what are termed the symptoms of poisoning by toxic drugs are alterations of cell-nutrition, and hence are diseases; short and violent ones in many instances, prolonged in others; for example, the chronic poisoning by ergot (see works on materia medica).

Of late years a new group of poisons has been discovered, which shows that toxication is widespread and common.

Organic poisons are formed by the action of bacteria (see next chapter) during the process of digestion, are absorbed, and give rise to toxic symptoms. Owing to the presence of specific bacteria, poisons are generated in the body, which, finding their way into the circulatory fluids, give rise to symptoms of poisoning.

The Influence of Anæmia.—The presence of oxidizable material and the removal of the oxidized material having been discussed, there remains another important factor—the oxidizer itself. Oxygen is received by the red blood-corpuscles in the capillaries of the lungs; it combines with the hemoglobin, an albuminate of iron, forming oxyhemoglobin, which in the capillaries throughout the body gives up its loosely combined oxygen to the tissues, is reduced, and takes up carbon dioxid, an excretory product. It is evident, therefore, that the volume of oxygen distributed throughout the body depends upon the amount of hemoglobin present—*i. e.*, upon the number of red corpuscles in the blood. A deficiency in this direction means lessened oxidation and its attendant effects.

Condition of the Circulatory System.—If, as a consequence of faulty action on the part of the heart or changes in the structure of the bloodvessels, the blood is not normally distributed, there is a resultant influence upon the distribution of nutritive material, and nutrition is disordered; for example, if as a consequence of disease of any of the heart-orifices the pressure of the blood in the vessels is lessened, changes in the nutritive balance result. If as a result of changes in the walls of vessels their elasticity is lessened, it is followed by faulty distribution of the food-supply. If any mechanical interference exist to the return-flow of the blood, stagnation with a retention of waste-products must follow.

The disease-causes thus far discussed have relation to effects upon the chemistry of tissue-life ; there remain other classes of disease-causes of great frequency. The first comprise all of those physical conditions or forces which act upon vital tissues and depress their activity or actually destroy them. They may be classed under the head of abnormal physical conditions.[1]

Abnormal Physical Conditions.—This class of disease-causes includes all injuries due to any of the physical forces. All surgical injuries may be placed under this head—diseases due to trauma. There are also included in the group diseases arising as a consequence of obstruction to ducts and to the natural outlets of the body. The actual causes of disease in these cases are secondary effects upon nutrition, arising out of faulty nutrition, and very frequently in consequence of retained waste-products.

Among the intrinsic causes of disease should be mentioned the influence of the nervous system. It is believed by some physiologists that there are special nerve-fibres whose function it is to preside over the nutrition of the parts of the body to which they are distributed ; these have been called trophic nerves. Unfortunately, the data upon which this belief is founded are too involved and too few to base positive opinions upon. It has been noted that when the Gasserian ganglion, the ganglionic centre of the great sensory nerve of the face, has been destroyed ulceration of the cornea has resulted, which has been attributed to the destruction of trophic nerves ; but when the eyeball is protected by stitching together the eyelids, so that there is no admission of foreign matters, ulceration does not occur. The more rational explanation, therefore, is that the ulceration is due to the loss of sensibility, the cornea not being able to recognize the presence of the foreign body. Again, injuries to nerves are followed by affections of parts to which the nerves are distributed. The classical example of this is that of Charcot ; the occurrence of acute bedsores in cases of certain degenerations of some portion of the central nervous system, which cause paralysis. Hyperemia, inflammation, and necrosis occur in rapid sequence over the hip or over the sacral region. The evidence is, however, insufficient to prove the presence of special trophic fibres. Many or most of the effects are perhaps more rationally explained by the theory of vasomotor influence upon nutrition. The calibre of the vessels is governed by special fibres, which by causing degrees of dilatation of the walls of vessels cause modifications of nutrition (see chapter on Vascular Disturbances).

Diseases are usually classified as general or local : the first class are those in which evidences of disturbance exist throughout the body at

[1] Green's *Pathology and Morbid Anatomy*, 8th ed.

large; the second, those in which the disease-manifestations are confined to some special region of the body. While this classification is useful in describing or naming distinguishing features between diseases, it is faulty in that it fails to describe the actual conditions existing. Some local diseases are manifestations of a general disease-process becoming evident in some one area; and, again, some general diseases are due primarily to disease which may be definitely localized in one region of the body. For example, in observing a typical case of gout it might be thought that the disease of the great toe was a local malady, when it is but the expression of a general cause acting locally; or, again, in observing the symptoms of typhoid fever it might be believed that the disease was a general one, which in one sense it is, but the primary cause is found in a bacterial ulceration of Peyer's patches in the small intestine, the constitutional symptoms being caused by the absorption and circulation of the poisons generated by the bacteria. Year by year the number of diseases traceable to a local source becomes greater, and the number classed as constitutional grows less.

CHAPTER III.

BACTERIOLOGY, WITH SPECIAL REFERENCE TO DENTAL PATHOLOGY AND THERAPEUTICS.

WITHIN the past twenty years a virtually new study has been introduced into medicine and surgery and their dependents, namely, that of Bacteriology. It is not to be understood that previous to this time what will be in the following pages studied as bacteria were unknown, but that it is only within that period that such modes of studying these organisms have been devised as render the study of direct practical use.

In 1683[1] Leeuwenhoek described bacterial forms obtained from scrapings of the human teeth. As early as 1762 Plenciz,[2] of Vienna, advanced the opinion that the bacterial forms, or animalcules as they were called, described by Leeuwenhoek and observed by himself, were the cause of all infectious diseases. These opinions did not receive general endorsement, and although the study of micro-organisms was pursued, it was not until some eighty years after that the germ-theory of disease received serious attention, when Henle taught the *modus operandi* of the bacterial origin of disease.[3]

The invaluable and immortal studies of Louis Pasteur demonstrated the physiology and physiological chemistry of bacteria, and showed conclusively the nature of fermentative processes.

Koch, through the devising of methods of isolating varieties of micro-organisms based upon their elaborately worked-out physiology, was the first to demonstrate clearly the relations between bacteria and infective diseases. Louis Pasteur had, however, previously studied and pointed out the relationship between plant and animal infectious diseases and bacteria, and had shown the modes of effectually combating such diseases.

Sir Joseph Lister's name has been so closely associated with that mode of surgical operating which deals with the freeing of wounds from bacteria, that modern antiseptic surgery is known as Listerism.

In order that bacteria as disease-causes may be studied systematically, the position of bacteria in the life-scale and their physiology must be examined into. It is noted, first, that their properties show them to belong to the vegetable kingdom, they occupying the lowest step of the

[1] Abbott's *Bacteriology*. [2] *Ibid.* [3] *Ibid.*

kingdom. All plants[1] are divided into two great classes, Phanerogamia and Cryptogamia. Phanerogams are those which flower and reproduce by seeds. Cryptogams are destitute of flowers, and reproduce through the medium of spores. Cryptogams are divided into two groups, those in which there is no distinction between leaf and stem, Thallophytes (Greek *thallos*, a young shoot, and *phuton*, a plant), and those which bear leaves, as many water plants.

The Thallophytes are subdivided into algæ, lichens, and fungi. The last-named order is that with which bacteria are classed. Fungi contain no chlorophyll, and hence are unable to derive their nutriment from inorganic material. Chlorophyll, the green coloring-matter of plants (*chloros*, green, and *phullon*, a leaf), is the substance through the agency of which carbon dioxid, CO_2, and ammonia, NH_3, are broken up and built anew into substances fit for the nutrition of plants. It is the substance which effects the decomposition of water and carbon dioxid and their recomposition into starch:

$$6CO_2 + 5H_2O = C_6H_{10}O_5 + O_{12}.$$

Fungi are, therefore, compelled to derive their nutritive material from existing organic compounds, which is, in fact, the reason why they become disease-causes. They are subdivided into four groups:[2] 1. Fission-fungi—Schizomycetes; 2. Mould or thread-fungi—Hyphomycetes; 3. Bud-fungi—Yeasts, Blastomycetes; 4. Animal fungi—Mycetozoa. All of these groups are interesting as throwing light upon many of the problems of pathology, and it is quite essential to comprehend the second class named in order to render clear the physiology of the first group, which is one of immediate and special interest to the pathologist.

Bacteria are vegetable organisms of extreme minuteness, many of them requiring high-power objectives and special staining-methods to make them visible. Owing to their methods of division they are termed fission-fungi, or the equivalent, schizomycetes, from Greek *schizo*, to split or rend, and *mukes*, a fungus. Like many other cells, they possess a cell-body and a cell-wall, and some of them a nucleus; their substance and cell-wall are composed of a modified protoplasm, called mycoprotein; the cell-wall of some is composed of cellulose. Some of them possess flagella, by which movement is effected. They are devoid of chlorophyll, and are thus unable to decompose carbon dioxid and water, and to effect the synthesis of starch from the elements of those compounds. To secure the carbon necessary to their life-processes they require to be brought in contact with solutions of carbohydrates. Their nitrogen is

[1] Thomé, *Structural and Physiological Botany.*

[2] Miller, *Micro-organisms of the Mouth.*

derived from albuminous substances with which they come in contact. Although these are the usual sources of the substances required for the life of bacteria, solutions of chemical substances may be made in which the growth of the organisms proceeds. The conditions necessary to their life and multiplication are, in general, the same as for all living bodies; they must have a proper food-supply, must be maintained at a suitable temperature, must be born or generated with normal vitality, and must have waste-products removed from in and about them. These factors vary within wide limits, as will be seen later. Their classification is attended with much difficulty; at present a classification based upon the form of the organisms appears to be as satisfactory as any. They are divided into three great groups—

1. The micrococci (*mikros*, and *kokkos*, a berry), including all of the spherical forms, or those having equal or nearly equal diameters.

Fig. 6.

a, staphylococci; *b*, streptococci; *c*, diplococci; *d*, tetrads; *e*, sarcinæ. (Abbott.)

2. The bacilli (*bacillum*, a rod), or rod-like forms; one diameter being greater than the other.

3. The spirillæ (Greek *spira*, a coil), or the curved forms.

All of these groups may be again subdivided. The micrococci are subdivided according to their modes of grouping. Double cocci are called diplococci (*diploös*, double). If in division the cocci arrange themselves in a line, they are called streptococci (*streptos*, a chain). If they agglomerate in bunches, they are termed staphylococci (*staphyle*, a grape). The organisms, upon reproduction, form groups which are called zoöglœa masses, the substance holding them together being formed by their cell-walls. The bacilli are of many and varied forms and differ as to their modes of grouping. They exhibit, in the same species, differences of form at different periods of their development. Many of them form spores

Fig. 7.

Zoöglœa of bacilli. (Abbott.)

(see Fig. 8). When brought under conditions unfavorable to their vitality "individuals themselves undergo alterations in their outline

FIG. 8.

a, bacillus subtilis with spores; *b*, bacillus anthracis with spores; *c*, clostridium with spores; *d*, bacillus of tetanus with end-spores. (Abbott.)

with the appearance of so-called involution-forms" (Abbott). The bacilli may again assume their typical forms when brought under conditions favorable to their development.

FIG. 9.

a, spirillum of Asiatic cholera (comma bacillus); *b*, involution-forms of this organism as seen in old cultures. (Abbott.)

Bacteria may be again divided, according to their mode of life, into the saprophytes (*sapros*, rotten, and *phuton*, a plant) and the parasites (*para*, near, and *siteo*, I nourish).

The saprophytic fungi are those which derive their nutrition from and multiply upon dead organic matter; they are the forms which break down dead animal matter from such complex molecules as albumin into the end-products—carbon dioxid, CO_2, ammonia, NH_3, and hydrogen sulfid, H_2S. Succeeding varieties of this group form from albuminous matters a series of substances having an increasing simplicity of composition. The saprophytic fungi play an important *role* in the economy of nature, as the products of the progressive decomposition cited serve as food for higher plants.

Parasitic bacteria are those which develop in the tissues of living animals and produce disease; they form the greater number of the group known as pathogenic organisms. All of the bacterial forms are to be found in this class. They gain access to the body through wounds, frequently large surgical wounds; by surfaces deprived of epithelium; a minute break in the continuity of the internal or external epithelial covering of the body may permit the entrance of the organisms to deeper tissues, where, under favorable conditions, they multiply and produce their specific effects. To generalize the effects of the

development of pathogenic organisms in the tissues: "At the point of multiplication, degenerations, necrosis, inflammations, and new growth of tissues, while the toxalbumins produced cause manifestations of poisoning. According to the variety of organisms, one or more of these results may predominate" (Ziegler). Many forms of organisms have points of election in which they find the most suitable soil for development, viz., the typhoid bacillus, in the glands of the ileum, Peyer's patches; the diphtheria bacillus, in the mucous surfaces of the pharynx and contiguous parts.

Many bacterial forms find a suitable habitat in the human mouth; the conditions of their food-supply and temperature invite the habitation and development of forms of all three of the groups cocci, bacilli, and spirilla, together with the characteristic leptothrix. This subject will be discussed in detail later.

In order to comprehend how bacteria act as pathogenic or disease-producing agents (*pathos*, disease, and *gennao*, I produce) an examination must be made into their physiology and physiological chemistry.

Fermentation.

Fermentation may be defined as "the series of changes which occur in solutions of organic substances exposed to the air, which result in radical changes in their chemical composition;" as, for example, the change of a solution of sugar into alcohol and carbon dioxid, and later into acetic acid, the formation of lactic acid in milk, the putrefaction of albuminous substances, and so on. The opinion that these changes are due to a simple oxidation may be entirely set aside. Although these decompositions are due to oxidation, hydration, and reduction, it has been clearly demonstrated that it is through the agency of fungi, and not mere contact with the oxygen of the atmosphere.

Sims Woodhead[1] comprehensively defines fermentation "as essentially the breaking up of chemical compounds, the *molecules* of which they are composed being separated from one another for a brief period, and then allowed to combine and form simpler and more stable compounds; owing to the setting free of such energy as has been stored up in the highly complex fermentable substance which is no longer required to maintain the high level of combination, a certain proportion of this energy is released in the form of heat; the temperature of a fermenting fluid is found to rise without the addition of any external heat."

If to a watery solution of sugar, or an infusion of vegetable juices containing sugar, a yeast (a bud-fungus, one of the blastomycetes) be added, and the fluid be maintained at a temperature of 35° C., it will be found that there is an evolution of gas, which, if passed into lime-

[1] *Bacteria and Their Products*, p. 87.

water, gives a precipitate—*i. e.*, the carbon dioxid has combined with the calcium of the lime-water to form calcium carbonate; the temperature of the solution rises above that of the surrounding atmosphere, and a test will show alcohol to be present in the solution and the sugar to have largely disappeared. Boiling the solution before adding the yeast does not prevent fermentation. Boiling the solution immediately after adding the yeast prevents fermentation—*i. e.*, boiling the fungus prevents its action, its vitality is destroyed. If a quantity of carbolic or salicylic acid, or mercuric chlorid, be added to the solution, fermentation does not occur. If the temperature of the solution be lowered, the process is much less active. If the access of free oxygen be prevented, fermentation does not occur, which illustrates that in fungi, as in all living cells, the vitality is lessened or destroyed by an insufficient food-supply and an unsuitable temperature; and, finally, it will be noticed that after a period fermentation ceases. If after a period some of the yeast be removed from the solution, it will be found that it has lost the power of inducing fermentative changes in other solutions in which it may be placed—it is dead: dead because of an accumulation of its waste-product, alcohol, about it. As with all vital cells, if their waste-products are not removed, the cells are smothered in their own excreta. This is the example of fermentation usually cited, but very many other types exist.

If to a solution of sugar a minute portion of the scrapings taken from a carious tooth-cavity be added, and the solution maintained at a temperature of about 35° C., it will be found, after a time, that the solution has acquired an acid reaction, and a chemical test will show that lactic acid has formed. A microscopic examination of the solution will demonstrate the presence of a large number of the fission-fungi forms. As each of these forms has probably its own peculiar physiology, several fermentations have been in progress, although lactic fermentation predominates. Miller[1] has shown that this fermentation ceases when the amount of acid present exceeds 0.75 per cent., this being the excess of waste-products which inhibits the vitality of the fungi.

These general principles govern the vital processes of all of the fungi, including the schizomycetes group, although the several varieties of bacteria have peculiar conditions of life which are qualifying clauses to the general proposition.

While it is shown that for the development of the yeast fungus and also for many of the bacterial forms the presence of free oxygen is essential, there are others which develop in the absence of free oxygen, and still others which develop either with or without the presence of free oxygen. Bacteria have, therefore, been classified by Pasteur into the aërobic, or those requiring free oxygen; anaërobic, those which

[1] *Micro-organisms of the Mouth.*

develop in its absence; and facultative-anaërobic, those which develop with or without the presence of free oxygen—or, 1st, obligatory aërobics; 2d, obligatory anaërobics; 3d, facultative-anaërobics.

A single species of bacteria rarely or never exists alone in a solution of organic matter, except as a result of special precaution upon the part of the investigator. Mixed cultures are the rule. When a number of species are introduced or are present some develop, others are quiescent; some find conditions favorable to their growth, others do not; but if the conditions be altered, a second or third variety prevails. Organisms may find in a field rendered, by an excess of waste-products, an improper culture-medium for one form of bacteria, a suitable medium in which to grow, so that progressive decomposition occurs in the medium. For example, in the successive stages of the breaking down of albuminous substances the compounds formed, while rendering the soil unfit for the life of the first invading organisms, fit it for the growth of other varieties, which effect further decompositions.

By processes of oxidation, reduction, and hydration many forms of bacteria effect a gradual decomposition of albuminous matter. Peptones,[1] substances having a less complex composition, are first formed; next, bodies of the compound ammonia type appear, the chemical and physiological equivalents of alkaloids derived from the higher plants, and hence called ptomaïns or animal alkaloids. Succeeding this, such nitrogenous bases as leucin, tyrosin, and the amins (methyl-, ethyl-, and propyl-amin) are formed. Next, fatty acids and such acids as butyric, lactic, and succinic, appear; next aromatic products, such as indol, phenol, and cresol, are formed; and the final decomposition is represented in the end-products—carbon dioxid, CO_2, hydrogen sulfid, H_2S, ammonia, NH_3, and water, H_2O.

Several forms of bacteria when grown in solutions of albumin produce substances of complex composition which act as poisons when introduced into the bodies of animals; these are termed toxalbumins. Many forms of bacteria when introduced in minute quantities into solid or gelatinous albuminous matter and kept at a proper temperature are found to increase in number; the gelatinous material around them becomes liquefied and undergoes decomposition, with the formation of substances which, introduced into living animals, act as poisons. If the solutions thus obtained be boiled and filtered, so that they are entirely free from the presence of the organisms which produced them, and then injected under the skin of an animal, evidences of widespread disorder are exhibited—the animal is poisoned. The effects will be governed, first, by the nature of the organisms producing the poison,

[1] Ziegler, *General Pathology*, 1895.

some producing more virulent substances than others; and, secondly, upon the size of the dose employed. If the effects pass away, the health of the animal is restored. If, on the contrary, the organisms themselves be introduced into the body, symptoms of poisoning appear and increase in severity, even though but few organisms be introduced. In some cases the symptoms persist until the death of the animal; in others they gradually disappear. If a portion of the tissue in which the organisms were introduced is used to inoculate other animals, similar symptoms appear in them. It is evident, therefore, that bacteria generate in the tissues substances which act as poisons.

Some species or forms exhibit a predilection for special tissues of the body; for example, a special bacillus develops in the glands of the ileum; others have a predilection for the large intestine, some for the spleen, others for portions of the nervous system, and so on; and possibly owing to the peculiar physiological chemistry of the part affected and to the species of bacteria, specific poisons are generated, which, taken into the circulation, produce characteristic symptoms.

Ferments.—The cells of the living body produce substances which have the power of transforming or changing the chemical composition of organic matter; for example, the epithelial cells of the salivary glands produce a substance which converts starch, $n C_6H_{10}O_5$, into glucose, $C_6H_{12}O_6$, a process of hydration. The cells of the peptic glands of the stomach produce a substance which changes albumin into peptone, probably also a hydration. The cells of the pancreas produce substances which cause the hydration of starch and of albumin and the formation of leucin and tyrosin. Such substances are known as ferments. They appear to take no part in the decompositions which they effect, and are called unformed ferments. The small plant-cells—bacteria—also give rise to bodies which effect similar changes, or the changes may occur in the cells themselves. It is through the agency of these latter substances, called "organized ferments," that the decompositions are effected which result in the chain of substances described under the decomposition of albumin. It is by reason of this property that bacteria act as disease-causes, and are capable of disturbing the physiology of the body. "When pathogenic fungi succeed in growing in the living body, if infection takes place, their action is in general characterized at the point of multiplication by degeneration, necrosis, inflammation, and a new growth of tissue, while the toxalbumins produced by them cause manifestations of poisoning."[1]

Many of the forms of bacteria, both saprophytic and parasitic, are found in ordinary drinking-water. Many of them, particularly the spores of bacilli, are found floating in the air; the soil to the

[1] Ziegler, *General Pathology*, 1895, p. 440.

depth of a metre (forty inches) contains numerous varieties. Above certain altitudes they are rarely found, and they are rare in the atmosphere of mid-ocean. At ordinary levels in temperate and tropical climates they are ubiquitous. They cling to most of the surfaces of bodies; are found on the skin and in its folds; insinuate themselves into the mouths of sweat and sebaceous glands, and swarm in the intestines and frequently in the mouths of animals. Among all of the varieties thus distributed, there are some which, gaining entrance to the tissues of the body, produce destructive diseases—*i. e.*, they are pathogenic. Organisms capable of inducing disease-conditions in the body are found in all three of the form-groups—cocci, bacilli, and spirilla. Many varieties of each of these classes have been shown to possess constant pathogenic features, and, further, they have been proved so constant attendants upon defined diseases that a causative relationship has been made out between the organisms and the disease. Special cultures of one variety introduced into the body produce always the same symptoms, and secondary cultures taken from the diseased organ induce the identical disease in other individuals.

A variety of coccus which when cultivated forms orange-colored colonies, and liquefies the gelatin upon which it is grown, when introduced into the body is followed by pus-formation—*i. e.*, it is pyogenic (from *puon*, pus, and *gennao*, I produce). From the manner in which the organisms collect into groups they are called staphylococci (from *staphyle*, a bunch of grapes); they are thus designated as the staphylococcus pyogenes aureus, the aureus from the orange color. Another pyogenic variety gives white colonies; hence it is called the staphylococcus pyogenes albus. Another, producing blue cultures, is called the staphylococcus pyogenes cyaneus. Still another, forming green colonies, is called the staphylococcus pyogenes viridis.

Several forms of bacilli have been shown to induce pus-formation; hence are called pyogenic bacilli.

A specific bacillus has been shown to produce Asiatic cholera, and from its form is called the comma bacillus. Another induces breaking down of tissue by the formation of tubercles, hence named the tubercle bacillus—the bacillus tuberculosis, the exciting cause of pulmonary phthisis and of diseases of the skin, joints, etc. To another variety of bacillus, typhoid fever is directly traceable. Numerous forms of cocci and bacilli when introduced into the tissues are followed by evidence of blood-poisoning, or septicæmia (Greek *septikos*, that which produces putrefaction, and *haima*, the blood). They are the bacilli or cocci of septicæmia. Several varieties found in the mouth are capable of causing these symptoms, and hence are known as the bacilli sputum septicæmia.

It is interesting and necessary to note that in many instances when

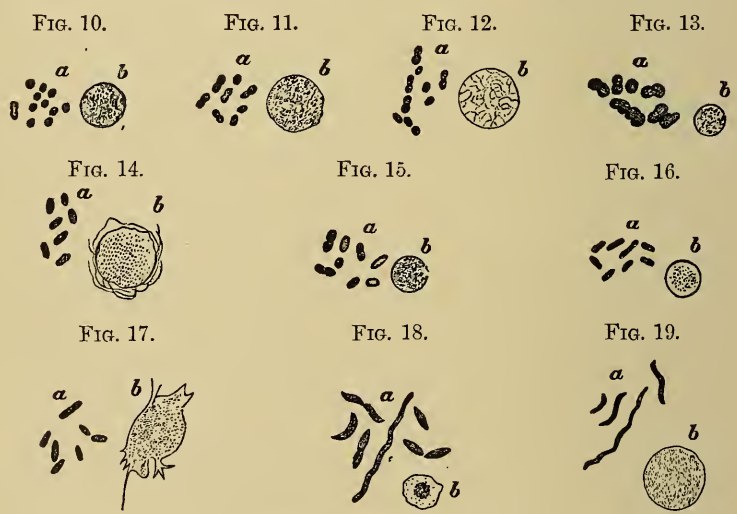
organisms are introduced into the body their action is self-limited, and in some cases a body once infected and recovered is insusceptible to future introductions of the same organism (see Immunity, Chapter II.).

Bacteria of the Mouth.

To the dental practitioner the forms of bacteria which are constant and those which are occasional inhabitants of the human mouth are of prime interest. Many of the saprophytic fungi are constantly present, finding in the food-débris, dead epithelium, abundant moisture, and suitable temperature, an extremely favorable soil in which to flourish.

The other two subdivisions of fungi, in addition to the schizomycetes, occasionally develop, as, for example, the bud-fungus (saccharomyces, oïdium albicans), which produces in neglected children the form of stomatitis called thrush.

Up to 1885 Miller[1] had isolated twenty-two different forms of bacteria from the human mouth. Ten of the twenty-two were cocci ; as in Figs. 10–13 ; some small, some very large. Five were short rods (Figs. 14, 15). A curved species, which liquefied gelatin and produced



Bacteria of the mouth. (Miller.)

a green coloring-matter, was called vibrio viridans (Fig. 18). Spirilla and the leptothrix were found. Of thirty species cultivated later, eighteen were cocci, eleven rods, and but one thread form.

"Of the twenty-two forms first described sixteen brought about an acid reaction when cultivated in beef-extract peptone sugar solutions. Several of the forms possess the power of converting sugar into lactic acid. In some cases the lactic fermentation is attended by the evolution

[1] *Micro-organisms of the Mouth.*

of gases, CO_2 and H, and if albumin be present H_2S may be generated; in others there is no formation of gases. There are a number of bacteria which almost invariably occur in every mouth."

Mouth bacteria proper:

1. Leptothrix innominata;
2. Bacillus buccalis maximus;
3. Leptothrix buccalis maxima;
4. Jodococcus vaginatus;
5. Spirillum sputigenum;
6. Spirochæte dentium (denticola).

These are all uncultivable, so that their physiology is not made out. Vignal found[1] among seventeen varieties of mouth bacteria the bacterium termo, bacillus subtilis, staphylococcus pyogenes aureus and albus.

In addition to the forms named, the following varieties of pathogenic fungi have been isolated from the mouth:[2]

Micrococcus of sputum septicæmia;
Micrococcus tetragenus;
Bacillus salivarius septicus;
Streptococcus septopyæmicus;
Micrococcus gingivæ pyogenes;
Bacterium gingivæ pyogenes;
Bacillus dentalis viridans;
Bacillus pulpæ pyogenes;
Pyogenic micrococci;
Actinomyces;
Saccharomyces albicans;
Spirillum sputigenum;
Pane's pneumococcus;
Bacillus saprogenes;
Streptococcus salivarius pyogenes;
Coccus salivarius septicus;
Micrococcus biskra;
Bacillus bronchitidis putridæ;
Bacillus tussis convulsivæ;
Bacillus pneumoniæ;
Bacillus pneumosepticus;
Pneumobacillus.

Of 111 mice in whose abdominal cavities human saliva was introduced, but 10 survived, the others dying in from fifteen hours to forty days, 42 of them within forty-eight hours.[3] In most of these cases the

[1] *Archives de Physiologie, norm. et pathol.*, 1886, No. 8.

[2] Miller. *Dental Cosmos*, Nov., 1891.

[3] *Ibid.*

micrococci of sputum septicæmia were found in a sero-purulent exudation in the peritoneal cavity. In 16 cases micrococcus tetragenus was present. In over 30 cases cocci, staphylococci, streptococci, and diplococci were present.

Miller divides the organisms into two classes, according to the mode of death of the animals attacked: first, those which produce speedy death through blood-poisoning with but comparatively slight local reaction; secondly, those which induce fatal pyogenic processes at the point of injection.

To recapitulate: the human mouth swarms with bacteria of the several classes; lactic, acetic, and butyric acid ferments; numerous saprophytic fungi; and organisms producing specific disease-conditions when introduced into the tissues. The pyogenic organisms are almost constantly present.

Phagocytosis.

The tissues of the body do not play an entirely passive part in the diseases caused by bacteria. While many tissue-cells of the body exhibit a resistance to and in some cases wage a warfare against invading organisms, the resistance is much more marked in some instances than in others. It had been observed, 1860–'70, by von Recklinghausen, Rindfleisch, Ponfincks, and others, that certain cells of the body took into themselves particles of dust and the material of disintegrated blood-corpuscles. In 1874 Ziegler observed the fibroblasts of granulation-tissue take up and destroy leucocytes—devour them. The wandering cells of the body, loaded with dust-particles and other foreign matter, appear upon the surface of mucous membrane.

In 1884 Metchnikoff[1] published the results of a series of studies made by him, principally upon the Daphnia, small fresh-water crustacea. He observed that when the spores of the monospora cuspidata,

Fig. 20.

1, a spore which has penetrated the intestinal wall and entered the abdominal cavity, where four leucocytes have surrounded its end: *m*, the muscular layer of the intestine; *e*, epithelial layer; *s*, the serous layer. 2, a spore surrounded by leucocytes from the abdominal cavity of a Daphne. (Metchnikoff.)

a bud-fungus, gained access to the alimentary canal of the animal it

[1] *Fortschritte der Med.*, Bd. ii. See *Comparative Pathology of Inflammation* (same author).

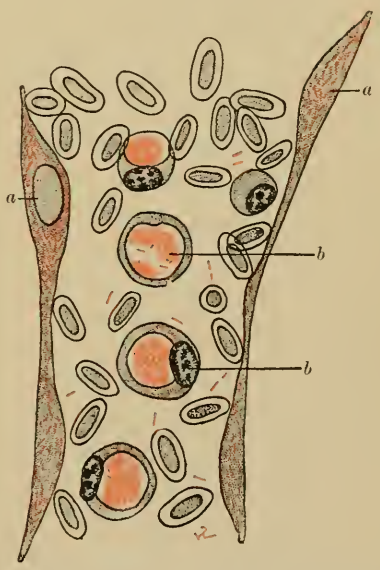
passed into the abdominal cavity through the intestinal walls. As soon as it entered the cavity it was attacked by leucocytes (Fig. 20), which took the spores into their bodies, and devoured and digested them.[1] "If the Daphnia is healthy, the monospora is gradually overcome; but if the animal be feeble or the monospora is ingested in very large quantities . . . the animal may eventually succumb to the attack."

The wandering cells of the body have also been shown to possess this power, which Metchnikoff has termed Phagocytosis—*phago*, I eat, and *cytos*, a bud (see Inflammation).

The reasons set forth by Metchnikoff, that this is Nature's means by which the living tissues defend themselves against invaders, and that these cells are specifically designed for this purpose, require modification. It is probably an expression of the nutritive function of simple cells. The process is not universal throughout the body, for in some instances the bacteria are taken up by the leucocytes, and in others they are left undisturbed.

Researches of Leber, Buchner, and others have shown that leucocytes may be repelled by certain chemical substances and attracted by others. This property of attraction and repulsion has been termed chemotaxis; positive when attraction exists, negative when there is repulsion. Buchner found that substances derived from certain bacteria, even in high dilution, possessed positive chemotactic properties, and that certain organic derivatives, such as methylamin, leucin, tyrosin, and urea, exhibit negative chemotaxis.[2]

Fig. 21.



Active phagocytosis. Endothelial cells enclosing the bacilli of swine septicæmia, from an hepatic vein of a pigeon: *a*, endothelial cells; *b*, leucocytes. (Metchnikoff.)

It is beyond question that phagocytosis plays an important part in the prevention of disease and the removal of foreign substances which gain access to the tissues, or are formed in the body. It is not the leucocytes alone which possess this power (Fig. 21). The endothelial cells of blood-vessels have this function, as have several other forms of mesodermal cells (Metchnikoff), notably the wandering corpuscles of connective tissue.

[1] Woodhead, *Bacteria and Their Products*, 1891; and Metchnikoff, *Ibid.*

[2] This subject is discussed at length in Ziegler's *General Pathology*, 1895.

Hugensehmidt[1] has shown that the filtered saliva of the human mouth, containing necessarily quantities of bacterial products, possesses marked positive chemotactic properties.

Nuttall[2] showed that the filtered serum of the blood possessed bactericidal power, and that degeneration of ingested bacteria occurred before the process of phagocytosis. Buchner found that the activity of the serum against bacteria was greater when deprived of its cellular elements. Boiling for half an hour destroyed the property. By dialysis or extreme dilution with distilled water its germicidal energy was checked; but if an equal dilution was made with sodium-chlorid solution (0.6–0.7 per cent.), the bactericidal power was not lost. Hankin,[3] Ogatta, and others have isolated from the spleen, lymphatic glands, and blood, globulins which in solution have germicidal power. Buchner has suggested that these germicidal substances be called "alexins." Noting the conditions under which serum germicides are observed, Vaughan[4] and also Halliburton[5] conclude that the substance to which the germicidal power is due is a nuclein.

[1] *Dental Cosmos*, vol. 38, p. 797 *et seq.*

[2] See Abbott's *Principles of Bacteriology*, p. 425.

[3] *Ibid.*

[4] *Ptomaïns and Leucomaïns.*

[5] *Chemical Phys. and Path.*

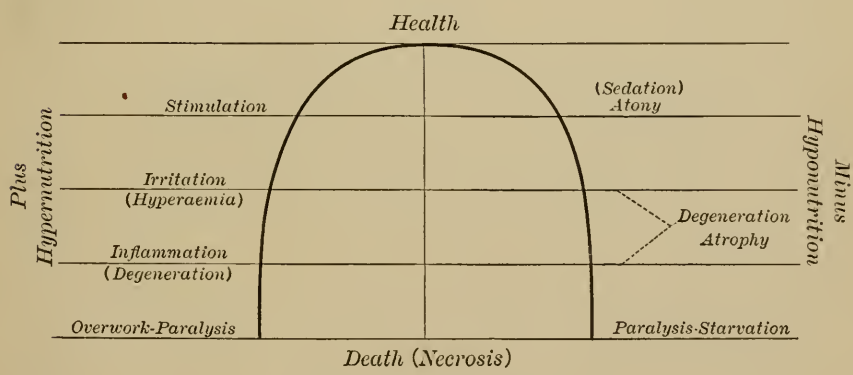
CHAPTER IV.

DISTURBANCES OF NUTRITION: ATROPHY, DEGENERATION, NECROSIS, HYPERTROPHY, TUMORS.

Owing to various disease-causes to which the tissues of the body may be subjected, they may suffer an alteration in the normal continuity of the cycle represented by development, growth, maintenance, and that gradual lessening of vitality which precedes the death of an individual. If there be such interference with the process of development that an organ is much below normal in size, the condition is spoken of as aplasia or agenesia. If the interference merely checks the growth of a developed organ, the condition is known as hypoplasia.[1] It is with disturbances arising during the period of maintenance that this chapter is concerned.

Disorders of nutrition are of two classes: in one class there is an excess of nutrition; in the other there is a deficiency. In either class disturbance in the nutritive equilibrium alters the vitality of tissue-cells. In one case, if stimulation be the process in operation, it is

Fig. 22.



but a question of time and degree until the death of the cells is brought about. In the other case, if sedation be the influence at work, it is also but a question of time and degree until the cells succumb. The extent of the degree of interference with nutrition, whether hyper- or hyponutrition, depends upon the extent of the influence in operation, the

[1] Ziegler, *General Pathology*, 1895.

effects ranging from slight disturbance of function to death of a part. The nature of these effects is graphically represented in Fig. 22. The two horizontal lines farthest separated represent health and death. Descending from the health-line to the death-line are the curved lines of a semi-ellipse, the path from health to death being a gradual, not a precipitate fall to death, in conditions of altered nutrition. It will be observed that these curved lines are crossed at intervals by straight horizontal lines, which represent stages in the vital descent, or grades of nutritive disturbance. If a general survey be taken of these several stages, based upon the observed phenomena of cell-life, deductions as to the nature of the alterations of physiology occurring may be made as illustrative examples.

Hypernutrition.

The effect of stimulation, first, upon the functions of organs, and, *pari passu*, upon their vascular supply (see Chapter V.), is represented by an increased nutrition. The irritability, contractility, and general functional activity of the part are increased. More food is appropriated, oxidation is increased, and hence more energy is set free and more work is done. Stimulation is therefore followed by an increase of functional activity. If the functions of the part be secretory, secretion is increased. Connective-tissue cells, whose function is the formation of basis-substances, form them in increased amount. Increase the stimulation, and the effect is one of irritation: vital processes become fretful, incomplete chemical changes occur in the cells, and functional activity is disordered. The energy developed in the cells of tissues manifests itself in nutritive, functional, or reproductive activities (Virchow), and hence, as the energy is developed in definite amount, if it be expended in one of these three directions, the other two must be correspondingly diminished. Thus irritation may be followed by increased secretion or functional activity, an altered metabolism in the cells; or the cells may exercise their reproductive function, in which case the number of cells is increased—*i. e.*, a part becomes hypertrophied.

HYPERTROPHY OR HYPERPLASIA.

By hypertrophy is meant an increase in the size of some organ, or portion of it, or of any tissues. Hypertrophy may be either simple or numerical. Simple, when the increase of volume is due to an increase in the size of cells; numerical, when due to an increase in the number of cells.

Causes.—Hypertrophy occurs when nutrition in a part exceeds the waste. In general terms, it is caused by a continued irritation of

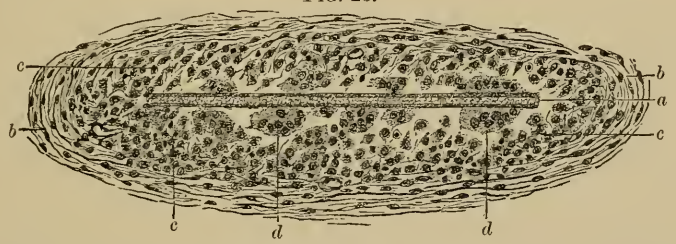
definite grade. It is, as a rule, intimately associated with a parallel increase in the local circulation of a part (see Chapter V. and Fig. 22).

Hyperplasia is frequently a reflex nutritive change due to an increased demand for work being made upon tissues; for example, when a muscle is repeatedly urged to an unusual amount of work short of marked fatigue, an increase in its function occurs, its capacity for work becomes greater, and if the strong stimulus (mild irritation) implied be continued, the cells increase in size, and, it may be, in number: all three phases of the expenditure of an increase of vital energy being represented—functional, nutritive, and reproductive. This is also represented in the reaction of the alternately acting and resting heart-muscles. If an unusual and continuous strain be placed upon the heart owing to an increase in the resistance it is normally called upon to overcome, an increase of the volume of the muscles follows.

Tissues or organs accustomed to performing work, if deprived of the work, use the developed vital energy in reproduction, and hypertrophy results. For example, upon the roots of teeth which have lost their antagonists the pericementum, deprived of its normal exercise, frequently forms hypertrophic growths of cementum upon the tooth-roots. When tissues accustomed to mechanical resistance due to their anatomical forms are freed from this resistance, they frequently hypertrophy in the direction of the accustomed resistance.

There is a form of cellular hypertrophy noted in some pathological states which should be mentioned in this connection, viz., under some

FIG. 23.



Dog's hair encapsulated in subcutaneous tissue: *a*, hair; *b*, fibrous tissue: *c*, proliferating granulation-tissue; *d*, giant cells. Preparation hardened in alcohol, stained with Bismark-brown, and mounted in Canada balsam. × 66. (Ziegler.)

forms or degrees of irritation, an increase in the size of cells may be noted characterized by incompleteness of formation; the nuclei of cells initiate the reproductive process, but it is incomplete. The nucleus divides, but the cell-body fails of division, the new nuclei subdivide, and the enclosing cell increases in volume, forming a giant or multinucleated cell. These cells appear where tissue is to be removed physiologically, as the roots of deciduous teeth. They are found about foreign bodies which have found entrance to tissues, about ligatures, dead tissues, etc.,

provided bacterial infection has not occurred and caused active inflammation. Their origin is probably from leucocytes and wandering tissue-cells, which, having undergone this peculiar variety of reproductive change, forming large multinucleated cells, and having their phagocytic property much increased, devour and remove many foreign substances.

It is probable that a similar condition exists about the root of an implanted tooth. The irritation caused by the presence of the foreign body causes cell-reproduction, complete in some cases, incomplete in others, the foreign substance being surrounded by a collection of embryonic cells. Some foreign bodies are devoured, others resist solution. In the first case, as soon as the foreign body is disposed of, the cells take on constructive, formative action, and are transformed into repair-tissue. In the second case, failing to remove the intruder, the cells may acquire a modified tolerance of it, and connective tissue may be developed around it—*i. e.*, it is encapsuled.

TUMOR-FORMATION.

Hypertrophy and hyperplasia are expressions of a degree of the reproductive function, physiological in character, having in the majority of cases well-defined causes, and serving an economic end in the body. There is another form in which there is an excessive activity of the cell-property, the reproduction being wholly malign in character, its products serving no physiological end; on the contrary, threatening life in the degree and rapidity of reproduction. Such reproductive forms are known as tumors. According to the etymology of the word, any swelling is a tumor (*tumeo*, I swell); but in its pathological sense the term is restricted to growths comprised under the following head: "A new formation of tissue possessing an atypical structure, not exercising any function or service to the body, and presenting no typical limit of growth."[1] This morbid reproductive process and consequent overgrowth may affect any of the tissues of the body. The growths may be divided into two great classes: first, those of the connective-tissue type—bone, cartilage, muscle, or ordinary connective tissue in any stage of its development; secondly, those of the epithelial type, arising from any epithelial structures—that is, any of the structures which arise from the epiblast or hypoblast of the germinal layers; the connective-tissue tumors are those composed of tissues derived from the mesoblastic layer.

Tumors may be clinically divided into two groups, according to their influence upon the life of the individual: first, benign tumors, or those whose growth is not a menace to life; secondly, malignant tumors, or

[1] Ziegler.

those which threaten the life of the individual affected. Malignant and benign tumors are found in both classes, connective tissue and epithelial.

The growth of a tumor is attended by a sapping of the vitality of a sufferer—the degree of the debility produced being apparently in direct ratio to the size and the rapidity of the growth. Besides the size and the rapidity of development of individual tumors, another element determines their malignancy, their position, and, furthermore, their occurrence in other parts, resulting in multiple tumor-formation. A tumor-victim acquires a peculiar appearance—a cachexia, whose intensity and rapidity of advance are directly dependent upon the degree of malignancy.

Tumors introduce no new form of tissue-element; they are reproductions of the cells of the tissues of the body. They may have the same cell-formation as the tissue from which they spring, and are then called homologous tumors; or they may have a different histological structure from the tissue in which they are found, being then called heterologous tumors. For example, a bony tumor growing from bone would be homologous; a cartilaginous tumor growing from gland-tissue would be heterologous.

Causes.—The causes of tumor-formation are unknown; it has been believed that their growth is due to parasites; this however has not been proved. A certain proportion of tumor-formations, 7–14 per cent.,[1] appear to be caused by traumatic injury; as, for example, in cases of mammary tumor a history of a blow or fall may be at times obtained.

Long-continued, sluggish inflammation appears to be causative of tumor-formation in an unknown percentage of cases. A chronic irritation of certain portions of the body, such as the junction between the mucous and skin surfaces of the lip, the sides of the tongue, etc., is a frequent antecedent to their formation. Ziegler gives a reasonable explanation of the origin of certain epithelial tumors in organs which are undergoing atrophy; for example, in advanced age the connective tissue of the body is undergoing atrophy and there is relaxation of its strata; the epithelium of the surface (or of glands), still possessed of its power of reproduction, proliferates and invades the connective tissue, producing cancer.

Tumor-formation consists in the reproduction of the cells of one or more tissues, and in the growth thus formed bloodvessels are developed. Tumors do not contain nerves.[2] Their blood-supply, however, is generous, so that for long periods a superabundance of nutritive material is carried to them; but after a variable period, depending upon the type of growth, the nutritive supply becomes disordered and degenerations occur.

About the more slowly developing tumors a condensation of connec-

[1] Ziegler.

[2] Green's *Pathology and Morbid Anatomy*, 8th ed.

tive tissue occurs in many cases, forming a distinct limiting wall or capsule from which the tumor may be enucleated.

The two great classes of tumors, those of mesoblastic and those of epi- and hypoblastic origin, may be subdivided into orders according to their histological peculiarities.

Class One.[1]

TUMORS OF MESOBLASTIC TISSUES:

Order One.

Tumors of mature connective tissue.

Bony tumors, or Osteoma.
Cartilaginous tumors, or Chondroma.
Fibrous tumors, or Fibroma.
Fatty tumors, or Lipoma.
Mucous tumors, or Myxoma.
Lymphoid-tissue tumors, or Lymphoma.

Order Two.

Tumors of the embryonic connective tissues:

The fleshy tumors, or Sarcoma.

Order Three.

Tumors of the higher tissues:

Tumors of muscle, or Myoma.
Tumors of nerves, or Neuroma.
Tumors of bloodvessels, or Angioma.
Tumors of lymphatic vessels, or Lymphangioma.

Class Two.

TUMORS OF EPIBLASTIC AND HYPOBLASTIC TISSUES:

Papilla of skin and of mucous membrane, or Papilloma.

Tumors of glandular tissue { Adenoma. / Carcinoma.

A separate class of tumors includes congenital mixed tumors, or Teratomata. The tumors of epiblastic and hypoblastic type are sometimes called Epitheliomata, for epithelial tissue is their characteristic histological structure.

Malignant tumors are found in both of the great classes, mesoblastic and epi- and hypoblastic. Carcinoma represents the type of malignancy

[1] Modified from Green's *Pathology*, p. 148.

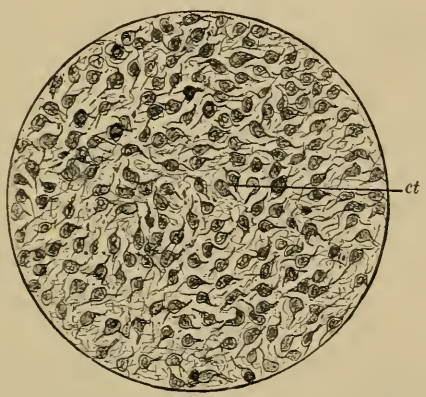
in the epithelial tumors. The sarcomata are the malignant tumors of the connective-tissue type.

Tumors are rarely composed of but one type of tissue; several types may be present, the tumor receiving its name from the tissue predominating. When the distinguishing feature of a tumor is two predominating tissues, the tumor is given a compound name; as, for example, when, in a sarcomatous growth, numerous large multinucleated cells characteristic of bone-marrow are found, it is called a myeloid sarcomatous tumor. When fibrous and sarcomatous tissues are distinguishing features the tumor is called a fibro-sarcoma.

Since the malignancy of a tumor is due primarily to the size and the rapidity of its growth, it is clear why sarcomata are more malignant than fibromata, and why some forms of sarcoma are more malignant than others. To illustrate:

Begin observation at the indifferent stage of connective-tissue development, when connective-tissue cells have first divided, reproduced;

FIG. 24.



Porcine embryo: *ct*, embryonic connective tissue of mesoblast. 2½ cm. × 250.

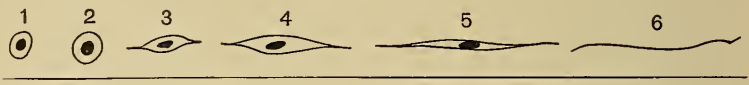
the tissue produced, seen in granulation-tissue and in the embryo, is at the indifferent stage, as seen in section of the embryonic jaw (Fig. 24).

Mesoblastic cells at this early period are in an indifferent stage; some of the cells shown in the figure will form bloodvessels, others will become bone-corpuscles, others will form fibrous and others muscular tissue. This structure has its analogue among tumors in a soft, fleshy, rapidly growing growth, called the round-celled sarcoma. As cells expend their vital energy in three ways (nutritive, functional, and reproductive activity), the embryonic cells of such a growth may expend their energy in nutrition (growth), and will then grow out of the indifferent stage into a more mature form of connective tissue, the ultimate form of one type being a fibre, an embryonic round cell undergoing a series of form-

changes from a small round cell to a long fibre (Fig. 25). The growth may cease at any stage of this form-change, the tumor composed of such cell-forms receiving a corresponding name. The embryonic connective-tissue tumors, as stated, are called sarcomas, the form of the cells composing them giving them a qualifying title.

In Fig. 25 are represented the stages of development of a connective-tissue fibre from a round cell. If growth cease at stage 1, and the

Fig. 25.



cell-energy thereafter expend itself in reproduction, a rapidly growing tumor composed of small round cells is formed—a small round-cell sarcoma, markedly malignant. If the cells expend a portion of energy in growth of cell-size, a large-cell sarcoma is formed, less malignant than the former. If the cells expend a portion of their energy in forming intercellular substance (reproduction), malignancy is less active. So the spindle-forms, 3 and 4, represent less rapid reproduction and lesser malignancy than 1 and 2, although the form 4, which should be of less rapid reproduction than 3, because of more mature organization, is frequently more malignant, because less intercellular substance is formed, as shown in Figs. 26 and 27, the energy represented in that process

Fig. 26.

Small spindle-celled sarcoma (from a tumor of the leg). × 200.

Fig. 27.

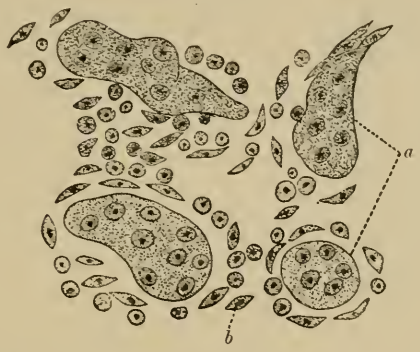
Large spindle-celled sarcoma. To the left the cells have been separated by teasing, so that their individual forms are apparent; to the right, they are in their natural state of apposition, such as would be seen in a thin section of the tumor. (Virchow.)

being used up in reproduction. The nearer the approach to the mature form, (6, Fig. 25), the slower the growth of the tumor, which, when composed of tissue of this type, loses its fleshy (sarcomatous) appearance and becomes fibrous, and is hence called fibroma.

When a sarcoma begins its growth from bone its histological character is frequently modified (Fig. 28). It contains large marrow-cells

which have undergone incomplete reproduction, forming giant multinucleated cells; this is a common form of tumor emerging from the sockets of teeth. Some of the cells of a sarcomatous growth may go on

FIG. 28.

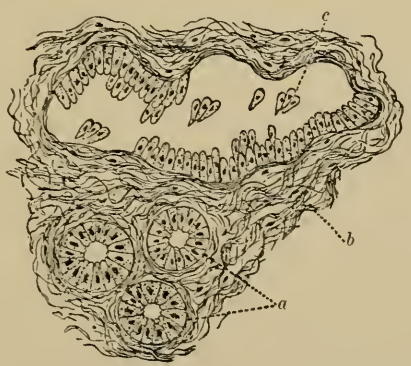


Myeloid epulis from lower jaw. *a*, multinucleated giant cells; *b*, oval cell. × 265. (Pepper.)

to maturity, while others remain at some stage of their developmental career. Malignancy will be modified according to the amount of mature tissue formed.

Epithelial Tumors.—Growths arising from epi- or hypoblastic tissues may be benign or malignant. What are called the adenomata may be taken as the type of the benign epithelioma; that is, comparatively benign. They present all of the characters of typical glandular tissue: numerous acini lined with epithelial cells and surrounded by connective

FIG. 29.



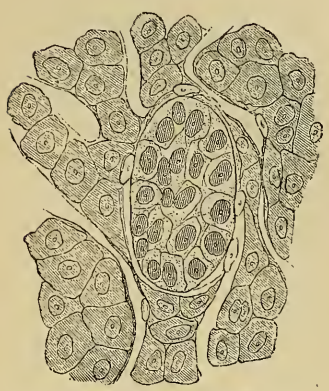
Adenoma of the breast: *a*, group of glandular acini; *b*, fibrous stroma; *c*, cells broken away from their attachment. × 265. (Pepper.)

tissue (Fig. 29). Tumors of this type may lose their comparative benignancy and become of the succeeding epithelial type.

Carcinomata.—These are growths arising from pre-existing epithelial

tissue, which possess the characteristics of epithelium developing without the limitations of a basement-membrane. Beginning upon a skin, or mucous surface, or in a gland, the reproduced epithelial cells are not sharply marked off from the connective tissue by a limiting membrane, but gaining entrance to the alveoli of connective tissue, they proliferate there, find their way into lymphatic vessels and lymphatic glands, and reproduce epithelial growths in such places of lodgement, so that a tumor having its origin in one part may give rise to tumors in other parts of the body (Fig. 30).

Fig. 30.



Section through an aggregation of very young cancer-cells, lodged like an embolus within a capillary of the liver. The parent growth was an adenocarcinoma of the stomach. Preparation stained with hæmatoxylin. ×300. (Ziegler.)

Like the connective-tissue tumors, types of carcinoma differ as to rapidity of growth in their original situation and in the degree of transference; these factors determine their malignancy. Tumors of the sarcoma group may also give rise to growths in other parts, the tumor-cells being carried thence by lymph-vessels or bloodvessels.

After a period, tumors frequently suffer such interference with their nutrition that degeneration occurs in them.

After removal, some varieties of tumors, both those which infiltrate surrounding tissues and those which are metastatic (or transferred from the original to other sites), show a tendency to recurrence; that is, removal does not effect a cure, and the tumor may upon reappearance assume another and a more malignant character.

Epithelial tumors never become tumors of the connective-tissue type; and, *vice versa*, connective-tissue tumors cannot become epithelial tumors. The distinction formed between epiblast and mesoblast in the embryo is maintained throughout life.

INFLAMMATORY DEGENERATION.

If the degree of irritation causing an increased activity of the reproductive function be exceeded, the entire nutritive balance of a part becomes disturbed in a violent manner; the vascular disturbance accompanying hypertrophic processes becomes much exaggerated in the tissues in the area involved. The nutrition of the cells of the part appears to be in abeyance, and vitality in them ceases; the protoplasm has lost its character, and in the affected area a collection of embryonic cells is found; this is the condition described in the next chapter under

the head of inflammation. The final stage of overstimulation has been reached—death by overwork.

Hyponutrition.

If the nutritive balance be disturbed upon the minus side, it amounts to the application of a sedative influence in varying degrees. As in the cases of hypernutrition, the stages or degrees of hyponutrition are intimately associated with the conditions of the vascular supply—its quality and its quantity. The first stage of hyponutrition is comprised in an atony, a lessened activity of the vital processes of a part. Cell chemistry is disordered, less oxidation occurs, hence a lessened heat-production; the function of the cell is diminished; if secretory, its secretion is lessened; if muscular, the cell has lessened contractility; the relations between nutrition and waste are disturbed; the part becomes physiologically wearied sooner than usual.

ATROPHY.

Causes.—If the disturbance in the nutritive supply be continued or be in more marked degree, the nutritive processes in cells become disordered, so that more cell-substance is broken down than is replaced by pabulum; that is, waste exceeds repair, and the part affected becomes diminished in size—*i. e.*, becomes atrophied. The atrophy may occur either because the food-supply is insufficient, or because the cell has undergone such change that it is not able to use its nutriment when present in proper quantity and quality. Atrophy is, however, more often traceable to an improper food-supply, or an excess of waste, than from any clearly made-out fault in tissues. A common source of insufficient food-supply is interference with the vascular supply to a part. It may be any of the causes (see Chapter V.) which lessen the size of the artery supplying a part.

Disuse of a part may be followed by a diminution of its vitality: it becomes atonic, and then atrophies. The atrophy is proportionate to the extent of the disease. Atrophy of organs may be a part of the cycle of life; as, for example, atrophy of the thymus gland in children, and atrophy of the mammary gland in the female after the menopause. Many atrophies of this type are properly termed resorptions; as, for example, in the thymus gland when the entire gland is removed. The loss of the roots of deciduous teeth and the loss of the alveolar process after teeth have been lost are examples of tissue-loss through resorption.

DEGENERATIONS.

If the nutritive balance be disturbed through intrinsic defect of cells, or through a further disturbance in the character or the amount of the

food-supply, the vitality of the cells undergoes more marked change. The character of the cell itself becomes altered, its chemical processes are markedly deranged, and changes in the cell-substance itself may be noted. The function of a part is impaired and soon ceases entirely. This is a cell-degeneration proper. Such processes are usually accompanied by shrinking in the size and diminution of the number of cells in a part; that is, degeneration and atrophy are processes commonly associated. In an actual degeneration the protoplasm itself becomes altered in character; the proteids of which it is composed are in part transformed into other substances.

There is another type of degeneration in which the protoplasm does not seem to have changed its chemical character, and yet foreign substances make their appearance in tissue and atrophy of its cellular elements follows.

True Degeneration.—The first type of cellular degeneration is that

FIG. 31.

Cloudy swelling of kidney epithelium: *a*, normal epithelium; *b*, epithelium beginning to be cloudy; *c*, advanced degeneration; *d*, cast-off degenerated epithelial cells. From a preparation which had been treated with ammonium chromate. × 600. (Ziegler.)

called cloudy swelling. The contents of a cell become markedly granular, hence the name of the condition, granular degeneration, and the cell increases in size. "This change is to be regarded as a disorganization of the cell-protoplasm following the absorption of liquid into its substance, and leading to a partial separation of its solid and liquid constituents. The cell may recover; but there is often complete destruc-

tion, the cell ultimately breaking down into finely granular fragments."[1] This is the parenchymatous degeneration found in continued fevers.

Fig. 32.

Muscular tissue of the heart (from a case of severe typhoid fever): the fibres are granular, the nuclei obscured, and the striation lost. × 400.

"It may be regarded as the first step toward fatty metamorphosis."[2]

Fatty Degeneration.—Fatty degeneration is a condition in which an accumulation of fat is found in the substance of cells as the result of partial decomposition of cell-substance itself.

Causes.—Fatty degeneration results from great disturbance in the nutritive balance of cells; that is, the cause of fatty degeneration is a high degree of disturbance of cell-nutrition. "The larger the amount of cell-albumin replaced by fat, the nearer is the whole cell to death."[3] Depressed vitality is always the proximate cause. It may be produced by improper food-supply and by changes in the vital powers of cells, or it may be an expression of the natural decadence of cells.

Disuse of a part which has normally an active physiological function is not infrequently followed by a degeneration of cell-substance and an associated atrophy of the part.

Mechanical interference with the blood-supply of a part is a prolific source of fatty degeneration of its cells. When the walls of vessels have undergone the changes known as atheroma and calcareous degeneration (which see), the arterial (food) supply to the tissues involved becomes much impeded. This change, occurring usually at a time of life when cell-vitality is on the wane, is followed by faulty metabolism in the cell; there is not that complete chemical interchange which occurs in normal metabolism, and degeneration occurs. When the coronary arteries of the heart become atheromatous fatty degeneration of the heart-muscle follows.

When the blood is deficient in quality, as in the several forms of anæmia, particularly pernicious anæmia, fatty degeneration and atrophy of organs may result in consequence of the faulty nutrition.

Fatty degeneration is one of the consequences of faulty oxidation in tissues, which explains its occurrence in the above-mentioned conditions, and it in turn lessens the oxidizing power of cells, the resulting cellular debility interfering with such processes as regeneration or the repair of tissues.

Fatty degeneration is the fate of nerve-fibres which have been severed from connection with their nerve-centre.

[1] Ziegler's *General Pathology*, 1895.

[2] Green's *Pathology and Morbid Anatomy*, 1895.

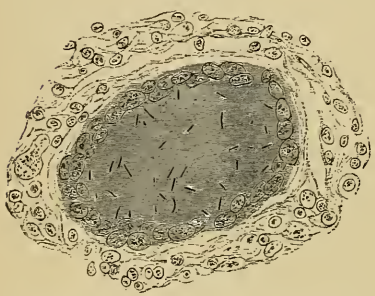
[3] *Ibid.*

In addition to the fatty metamorphosis which occurs in tissue-cells as the result of faulty metabolism, this change of cell-albumin also occurs in other pathological states; for example, the cells in certain inflammatory effusions undergo fatty metamorphosis.

The process occurs in the cells of tumors when the growth of their vascular supply does not keep pace with the volume of the tumor.

A peculiar change sometimes takes place in portions of tissue which have suffered fatty degeneration, the change affecting particularly parts which have degenerated in consequence of the presence of the bacillus tuberculosis. The presence and proliferation of the bacillus in tissues appear to be attended by phagocytic action (see Chapter III.) upon the part of the cells about them. The irritation resulting from the action of the bacillus causes incomplete reproductive activity in the cells, and multinucleated cells are formed. The bacilli are taken into the bodies of these cells, the irritation set up causing (Fig. 33) an emigration of leucocytes from the adjacent vessels which occupy the connective-tissue spaces; the accompanying effusion solidifies, and in the affected spot is formed what is termed a tubercle. The cells after a period may undergo fatty degeneration, and following this the mass is transformed into a cheesy substance, in consequence of which the process is called caseation.

FIG. 33.



Tubercle bacilli in giant-cell (from tuberculosis of horse). ×600. (Cheyne.)

In apoplectic effusions into the brain-substance the effused blood and part affected undergo fatty degeneration and caseation.

It not infrequently happens that in these caseous masses calcium salts are deposited. At times the entire mass may, from some unknown cause, excite active inflammation.

FATTY DEGENERATION OF VESSELS.—The occurrence of fatty degeneration of the heart-muscle due to obstruction in the coronary arteries has been alluded to. A similar condition may occur in the muscle-fibres of the middle coat of bloodvessels. It occurs most frequently in the internal, the endothelial coat, or the tunica intima of vessels. Droplets of fat are formed in the endothelial cells lining the vessels. "In the smaller arteries the fatty degeneration is more liable to affect the external coat."[1] In some forms of nephritis (Bright's disease) fatty degeneration affects the endothelial cells of the capillary vessels, leading to their rupture.

[1] Green's *Pathology and Morbid Anatomy.*

Mucoid, Colloid, and Hyaline Degeneration.—The albumin of cells may undergo other chemical changes than transformation into fatty substances; they may undergo mucoid, colloid, or hyaline transformation. The causes of the degeneration are not made out. The function of the part affected is destroyed.

Lardaceous Degeneration.—This type of degeneration is known as amyloid, albuminous, or waxy. The formation of the material from which this condition derives its name is preceded by an unknown type of degeneration of the cells of the part affected. The degenerative processes appear to be the result of long-continued suppuration due usually to tubercular diseases. In the connective tissue about the degenerated cells a substance akin to albumin is deposited, which causes swelling and a pseudo-hypertrophy of the organ affected. The substance gives a reaction with iodin resembling that of starch, hence the name amyloid (*amylum*, starch). It may affect any organ of the body. It usually appears first in the connective tissue lying between the inner and middle coats of small arteries. The swelling caused by the infiltration markedly lessens the calibre of the vessels and diminishes the nutritive supply of the parts supplied by the artery, which may lead to fatty degeneration and atrophy of the insufficiently nourished parts.

Calcareous Degeneration.—This condition must be clearly distinguished from ossific or bone-forming changes which occur in tissues. In bone-formation there is a constructive process in which cells build a definite type of formed material. In what is called calcareous degeneration there is a deposit of calcium salts, mainly the phosphate and carbonate. In bone-formation the lime salts are combined in a definite manner with an albuminous basis (see Calco-globulin). The phosphates and carbonates of calcium and the carbonate of magnesium are held in solution in the serum of the blood by virtue of the carbon dioxid in the blood. It is believed that the deposit of calcium salts in a tissue is more than a mere precipitation, and that calcification results from the combination of the salts with an albuminous base and with fatty acids. These deposits occur in tissues which are in a degenerative or dying state. They follow frequently as a secondary state upon fatty degeneration, caseation, and hyaloid degeneration of tissues, particularly of connective tissues. A similar deposit occurs in the affected joints in gout, when sodium biurate is deposited in the affected tissues. This is particularly notable in the walls of bloodvessels which have suffered previous degeneration.

The changes which pronounced degeneration or death of cells brings about in tissues appear to create an affinity between such substances and the calcium salts of the blood, so that they form intimate compounds.

The deposits may be in the cells themselves, as well as in the intercellular substance. They affect particularly the white fibrous connective tissues. What appears to be a cardinal principle in these calcic deposits, is the formation of substances during or following degenerative changes in parts having a sluggish circulation, which substance renders insoluble the calcium salts held in solution by the carbon dioxid of the blood, or possess a stronger chemical affinity for the salts than the carbon dioxid which holds them in solution.

Calcification of Arteries.—In or during the series of nutritional disturbances associated with senility, the degeneration of tissues accompanying old age, calcareous deposits may occur in the middle coats of arteries, transforming these normally elastic tubes into rigid tubes of lessened calibre. Such arteries cannot perform their normal function in regulating the blood-current, and parts supplied by them suffer more or less nutritive disturbance and in some cases actual death. Calcareous areas frequently form in such portions of arteries as have suffered from an inflammatory degeneration of the tissues of the deeper layer of the inner arterial tunic (*i. e.*, atheroma).

NECROSIS.

When the depression of vital activities due to disturbance of the nutritive balance becomes more marked than those grades productive of atrophy and degeneration of a part, the vital processes of the cell are paralyzed—it dies from starvation, the condition being called necrosis, from *nekros*, dead.

Causes.—The conditions which bring about a cessation of vitality in the cells of tissues may be grouped under two heads:[1] 1st. Interference with the supply of nutritive material; 2d. Destruction of the vital activity of cellular elements.

Class I.—Necrosis arising out of the first group is caused by obstruction of the vascular supply, through occlusion of arteries, veins, or capillaries.

1st. Obstruction of the arteries. If from any cause—surgical ligation of an artery, pressure upon it by effusions or new growths, degeneration or affections of the arterial walls, the presence of an embolus or thrombus (Chapter V.)—the flow of blood to a part is arrested, the nutritive supply ceases and the cells dependent upon that vessel perish. If the part receive a collateral arterial supply, the cells may retain their vitality, although if this supply be inadequate they are in danger of degeneration and atrophy. This will explain the greater relative frequency of extensive necrosis of the lower jaw, as compared with necrosis affecting the upper jaw, the lower jaw being supplied mainly by one

[1] Green.

large arterial trunk, while in the upper jaw there is a freely anastomosing circulation.

2d. Obstruction of the veins. If the entire venous outlet of a part be obstructed, there is not that removal of waste-products necessary to the life of cells; moreover, access of nutritive material is prevented and the parts die.

3d. Obstruction of the capillaries. Complete obstruction of the capillary supply to a part is followed necessarily by a cessation of nutrition in the part; consequently necrosis results. For example, when an inflammatory effusion occurs between the surface of a bone and the periosteum, the capillaries are torn from their attachment; and if the condition be prolonged, necrosis of the underlying bone results. When the effusion occurs outside the periosteum its pressure may cause occlusion of the capillaries of the part. The interference with the nutritive supply may be due to a lack of force with which the blood is propelled, owing to insufficient action of the heart. Necrosis is not infrequently due to the violence and continuance of the inflammatory process in a part. Coagulation of the blood in the capillaries of a part occludes the circulation and death results.

Class II.—Destruction of the vital activities of cells may be caused by any of the physical forces or by the action of chemical agents, including among the latter the poisonous substances formed through the action of bacteria.

Injuries, blows, excessive heat or cold, the passage of powerful electric currents, are all influences which directly injure or permanently destroy vital activities of cells. The application of chemical agents which so act upon cell-substance as to change its character produces necrosis. While this is particularly true of such substances as powerful acids and alkalies, which immediately destroy cell-integrity, it is also true of milder agents acting for longer periods. Certain poisons, particularly those of bacterial origin, paralyze the vital activities of cells and necrosis results.

The occurrence or non-occurrence or the liability to necrosis will largely depend upon the degree of vital energy of cells prior to the action of the active causes of necrosis. Parts debilitated from any cause are more liable to necrosis than those which have suffered no debility. A part chronically ill-nourished subjected to the causes producing degenerations is liable to suffer necrotic changes, for the several degenerations and atrophy, as shown in Fig. 22, are but successive stages leading to necrosis.

When a tissue undergoes death as the result of the infliction of an injury the process is called necrosis *per se*, to distinguish it from the

form of death which occurs in a descending scale, which process is called necrobiosis.

A necrosed part acts as an irritant to the tissues about it, inaugurating an inflammatory reaction which marks off the dead from the living parts. The dead part is sequestred, and hence is called a sequestrum.

Coagulation-necrosis. — When a dead tissue contains coagulable material and the necessary ferments (Chapter V.) the parts undergo coagulation. The cells and parts about become solidified; the cells lose their nuclei and do not stain as usual.

Liquefaction-necrosis. — When the necrosed parts are saturated with lymph the dead part breaks down and liquefies.

Dry Gangrene.—When parts have died as the result of stoppage of circulation and the access of fluids is prevented, the dead part may undergo a species of mummification.

Moist Gangrene.—When the organisms of putrefactive fermentation gain access to a necrotic part they break down the dead tissues, with the formation of products described in Chapter III. The formation of such gases as hydrogen sulfid, H_2S, hydrogen phosphid, PH_3, and ammonium sulfid, $(NH_4)_2S$, gives the offensive odor to gangrenous parts. The amount of water necessary for the development of putrefactive organisms is not present in dry gangrene, hence putrefactive decomposition is absent or long delayed in this condition.

CHAPTER V.

DISTURBANCES OF THE VASCULAR SYSTEM.

Ever since the physical nature of the circulatory apparatus was pointed out by William Harvey in 1628, disturbance of the equilibrium of the circulation has been held to have a close relationship with the process of nutrition. The sum and substance of contemporary view of this matter is that an increase in the flow of blood to a part means an increase of nutritive material in the part; that is, increased circulation means hypernutrition. We shall see, however, that this belief can only receive qualified acceptance in the light of the latest views of this branch of physiology. A decrease in the supply to a part is followed by a diminution of nutritive material, and, as a consequence, hyponutrition prevails. This distinction at once divides local disturbances of the circulation into two groups, viz., hyperæmia, a condition in which there is an excess of blood in a part; and anæmia, a condition in which there is a deficiency of blood in a part. In this division, however, the latter term is used inaccurately, for anæmia has come to be applied to those conditions in which there is a deficiency of red corpuscles in the blood, or to those conditions consequent upon hemorrhage, in which the general volume of the blood is lessened in amount. The corresponding term, indicating an excess in the volume of the circulatory fluids, is plethora. The word ischæmia, from the Greek *ischo*, I stop, is used to express the condition opposite to that of hyperæmia.

Condition of the Blood.

There are two elements which enter into the discussion of the subject of the relationship existing between nutrition and circulation, the first being the composition of the blood, and, secondly, its mode of distribution—its equable supply to the several tissues and organs of the body. The first is a factor of prime importance, irrespective of the second, for abnormalities in the composition of the blood, the vehicle for nutritive material, and of what is of almost equal importance, the waste-products formed in the body, are inevitable precursors of disturbances of the normal nutrition of tissues—*i. e.*, are productive of disease.

The first essential to the proper functionating of the great chemical

laboratory, the human body, is that it shall receive material which will enable it to continue its great variety of chemical operations. The first of these materials, in point of importance, is oxygen ; for, as pointed out by the physiologist, vital action is largely dependent upon the process of oxidation.

In the absence of oxygen the protoplasm of cells, no matter how differentiated, whether a neuron of the brain, a parenchymatous cell of the liver, an epithelial cell of the kidney, or the odontoblast of a dental pulp, cannot be the seat of chemical reactions and reductions which transform nutritive matter into protoplasm, or which reduce the waste-products of cellular activity to substances which may be removed from the cell. Oxygen, therefore, is essential, not only to the reconstructive action, constructive metamorphosis, but also to the removal of waste-products, which, if remaining unoxidized, literally poison the protoplasm with which they remain in contact.

Changes in the Composition of the Blood.—The first factor to be studied, therefore, in connection with the composition of the blood is its oxygen capacity—in short, the number of its red corpuscles. It is usually stated that the average number of red corpuscles in human blood is 5,000,000 in a cubic millimetre.[1] This proportion is temporarily altered by divers influences, so that a deficiency of red corpuscles is only spoken of when the deficiency is persistent. When the proportion is lessened the condition is spoken of as oligocythæmia (Greek *oligos*, few). The special constituent of the corpuscle with which oxygen-carrying is associated is hæmoglobin, chemically an albuminate of iron, which holds oxygen with a light affinity which carbon dioxid overcomes, and displaces the oxygen. If hæmoglobin be deficient in amount, or if there be such a pulmonary condition that a free interchange between the blood in the capillaries of the lungs and the air in the acini is, interfered with, it is evident that the supply of oxygen to the tissues will be deficient, and a condition of suboxidation will exist throughout the body, nutrition will be imperfect, and waste-products of incomplete metabolism be formed, with a tendency to their retention. Arterial blood containing those substances fitted for nutrition becomes unfitted for this office if the processes of digestion, absorption, and glandular functions, particularly those of the liver, do not properly transform food-stuffs into substances fitted for the proper performance of cell-function. Those miniature chemical laboratories, living-cells, require for their special chemistry, substances elaborated by the great laboratories of the digestive system. If these substances are not formed, the cells throughout the body receive material unfit for their purposes. The peculiar chemistry of the several organs of the body will modifiy the substances contained in, particularly, the

[1] Foster's *Physiology*, 5th ed.

venous blood of any region, so that the composition of the blood varies according to the organ in which it is found. In general terms, arterial blood contains the nutritive materials of the body, and the veins the waste-products; to this rule, however, there are marked exceptions; for example, the blood of the renal artery contains waste-matters (urea, etc.) to be eliminated, and the blood in the hepatic veins contains the food-substances which have been prepared by the liver.

If, owing to continued faulty physiology, the quality of the blood be altered, either by not receiving properly elaborated food-materials or from the presence of an undue amount of substances which should be removed as waste-products, the nutrition of organs suffers, and there is inaugurated the series of disturbances described in Chapter IV. Circulating in the body the waste-products of tissue-metabolism, if present in undue amount, act as irritating or paralyzing agencies upon the vital activities of cells, particularly upon cells whose activities are below normal, hence they are productive of degenerations. Urea and uric acid are marked examples of such irritants. If the elimination of urea be checked, owing to disease of the kidneys, the soluble urea circulating in the blood-current acts as a poison to the organs of the body, and evidences of widespread functional disturbance make their appearance. Uric acid in the form of soluble urates of sodium, which should be eliminated by the kidneys, if retained in undue amount, owing to kidney-disease, is precipitated in regions having a sluggish circulation, where the vitality of the cells has been diminished or degenerations are in progress. It, in its turn, may act as an irritant in parts in which it is deposited.

Substances formed in the intestinal canal as the result of faulty intestinal digestion, and poisons developed there through the action of bacteria, may be absorbed, and if that destroyer of poisons, the liver,[1] fails to neutralize them, they gain entrance to the general circulation and act as poisons (see Septicæmia and Pyæmia).

Coagulation of the Blood.

The blood contains a substance, probably a globulin, out of which fibrin is formed; hence it is termed fibrinogen. It is permanently soluble in the alkaline blood-serum under normal conditions; but when from any cause injury or degeneration of white blood-corpuscles occurs, substances, called ferments, are set free from the corpuscles, which in the presence of calcium salts (present in the blood as phosphates and carbonates) combine with fibrinogen to form a new compound, whose solubility is altered, and a new and solid substance makes its appearance—fibrin. This reaction occurs in blood removed from the body, constituting what is called the coagulation of blood. When

[1] Brunton, *Pharmacology and Therapeutics*, 3d ed.

blood is drawn in a vessel, after a period it is noted that it changes from a fluid to a jelly-like mass, red in color. The conditions under which the blood is placed cause partial disintegration of leucocytes, paraglobulin and ferments are set free, which, combining with the fibrinogen of the blood, form fibrin; the corpuscular elements become entangled in the meshes of the fibrin.[1] Under some conditions this process occurs in living bloodvessels. When from any cause the endothelial cells lining bloodvessels have suffered injury, whether by mechanical injury or the presence of substances which cause irritation, an accumulation of leucocytes occurs in the locality and the reaction called coagulation occurs. The process seems to be determined by a slowing of the blood-current in the affected vessel, and it is extremely probable that changes in the chemical composition of the blood play an important part.

Thrombus.—When a coagulum forms in a vessel it is known as a thrombus (Greek *thrombos*, a clot of blood). It may form in the vessels or in the heart; may remain where it has formed, or be transported to other parts, forming what is called an embolus (Greek *embolon*, a piston). Whatever its situation, the significance of a thrombus or embolus is mechanical interference with the circulation, and its effects are governed by the extent to which the blood-supply of a part is occluded. If the embolus be formed of tumor-cells or of bacteria which have gained entrance to the circulation, secondary disease-processes are set up. Remaining in the situations in which they were formed, thrombi undergo degenerative changes. In one of the typical situations, in varicose vessels, when the vascular current is much slowed, thrombi frequently form and undergo calcareous degeneration (which see), forming what are called phleboliths (vein-stones). If the occluded vessel be what is called a terminal artery—that is, an artery whose branches spread like those of a tree, without anastomosis—the area to which it is distributed undergoes degeneration and death. The backward pressure from the veins upon the blood-current of the capillaries causes rupture of the latter, and an extravasation of blood into the wedge-shaped (Fig. 34) area occurs, forming what is called a hemorrhagic infarct.

FIG. 34.

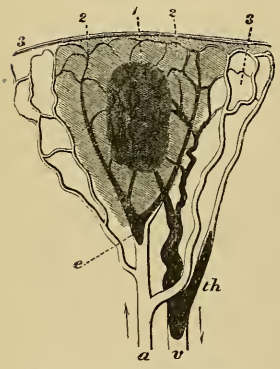
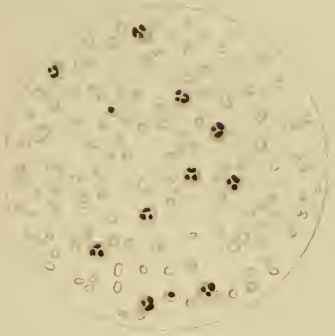


Diagram of a hemorrhagic infarct: *a*, artery obliterated by an embolus (*e*); *v*, vein filled with a secondary thrombus (*th*); 1, centre of infarct, which is becoming disintegrated; 2, area of extravasation; 3, area of collateral hyperæmia. (O. Weber.)

[1] For extended description of this process see Foster's *Physiology* and Landois' *Physiology*.

PLATE I.

Fig. 1.

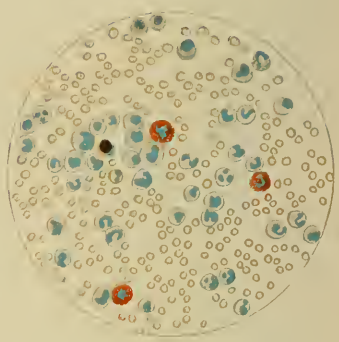


Severe Aæmia with Leucocytosis.

Dry preparation. Fixed with picric acid. Stained with hæmatoxylin Böhmer, x 300.

Red corpuscles few, almost colorless, varying in size, show poikilocytosis; two nucleated reds (normoblasts). The increase in the white cells seen to be in the polynuclear elements. (Rieder's "*Atlas der Klinischen Mikroskopie des Blutes.*")

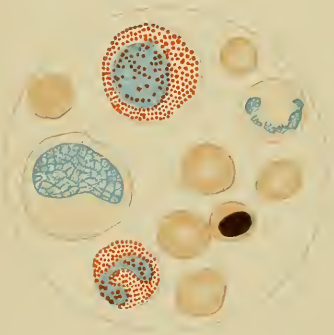
Fig. 2.



Splenic-myelogenic Leukæmia.

Eosin-hæmatoxylin, x 300. Red corpuscles rosy-red, of nearly uniform size, round. To the left a normoblast with eccentrically placed nucleus. Many large mononuclear leucocytes (myelocytes) and three eosinophiles seen. (Rieder.)

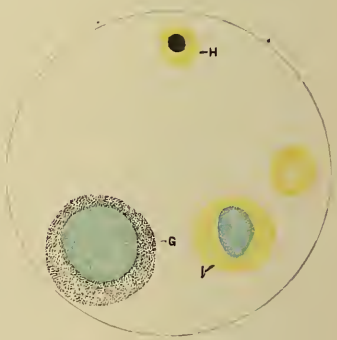
Fig. 3.



Splenic-myelogenic Leukæmia.

Same case. Eosin-hæmatoxylin, x 1100. One normoblast, one polynuclear leucocyte, one myelocyte, two eosinophiles. The neutrophilic granules of the polynuclear leucocyte and of the myelocyte do not show with this stain. The large mononuclear eosinophile above is believed to be also a myelocyte (Markzelle), the smaller one below, an eosinophile such as can be found in normal blood. (Rieder.)

Fig. 4.



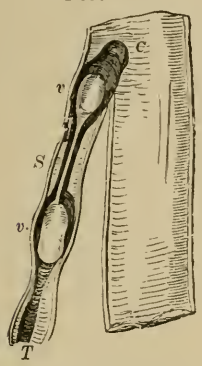
Myelocyte, normoblast, megaloblast.

Triple stain. G, Myelocyte showing neutrophilic granules; H, normoblast, both from a case of splenic myelogenic leukæmia; I, large nucleated red corpucle (megaloblast) from a case of pernicious anæmia. (Osler.)

The death of the dental pulp is no doubt frequently due to this process.

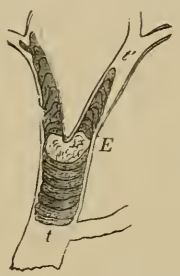
Thrombi formed in veins may find their way, *via* larger veins (Fig. 35), into the heart, and thence be driven into the branches of the pulmonary artery. Thrombi formed upon the arterial side, if detached, are driven along the artery to a vessel or junction of vessels refusing passage to them, when the results of thrombus are suddenly set up (Fig. 36).

FIG. 35.



A thrombus in the saphenous vein, showing the projection of the conical end of the thrombus into the femoral vessel: *S*, saphenous vein; *T*, thrombus; *C*, conical end projecting into femoral vein. At *v*, *v*, opposite the valves, the thrombus is softened. (Virchow.)

FIG. 36.



Embolus impacted at the bifurcation of a branch of the pulmonary artery, showing the formation of thrombi behind and in front of it, and the extension of these as far as the entrance of the next collateral vessels: *E*, embolus; *t*, *t'*, secondary thrombi. (Virchow.)

PHAGOCYTES OF THE BLOOD.

The white corpuscles, the leucocytes of the blood, are of great clinical importance. Several varieties of the white corpuscles are to be recognized:[1] 1. Lymphocytes, derived from the lymphoid tissues of the body, forming 20 to 30 per cent. of all of the leucocytes of the blood; they are probably immature leucocytes. 2. Large leucocytes having a single nucleus, which take up acid eosin, stain readily (eosinophiles), forming 2 to 3 per cent. of leucocytes of blood. 3. Smaller leucocytes containing several nuclei, which stain only with a mixture of basic and acid dyes, and hence called neutrophiles; they form two-thirds the entire number of leucocytes. 4. Transitional forms, constituting about 3 per cent.; they are between the mononuclear and polynuclear forms. 5. Eosinophile cells, size of No. 3 nuclei, variable; they constitute from 2 to 4 per cent. of leucocytes. (Plate I.)

Under some conditions, notably during diseases in which suppura-

[1] Park's *Surgery*, vol. i., and Metchnikoff's *Lectures*.

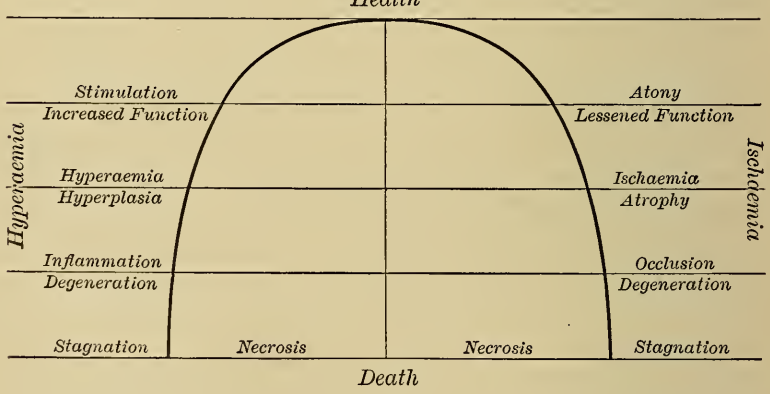
tion occurs, there is a marked increase in the polynucleated leucocytes. This fact has been applied as a diagnostic sign of the existence of suppuration;[1] if leucocytosis be present during the course of surgical diseases, suppuration and retention of pus are to be suspected. The large mononuclear and the polynuclear forms of leucocytes have pronounced phagocytic activity; it is held that the reason for their increase is the presence in some part of the body of substances acting as tissue-irritants. These cells are most active in disposing of invading bacteria, and are present in great numbers in areas of inflammation.

Disturbances of the Vascular Mechanism.

As stated, disturbances in the equilibrium of the circulation may be conveniently grouped under two heads, a hypo- and a hyper- group, or conditions of ischæmia and conditions of hyperæmia. These may be again divided according to the extent of the disturbance, ranging, on the one hand, from a slight decrease in the circulation of a part, to its entire occlusion or stoppage, on the other hand: the range extends from a slight increase of circulation to an entire paralysis of vessel-walls. In either event the tissues depending upon the supply of blood through the affected vessels are adversely affected, it may be to the extent of their death.

The grades and effects of both types of vascular disturbances are graphically represented in Fig. 37.

Fig. 37.



The cause of the lessened blood-supply to an organ may be at any portion of the circulatory apparatus—the heart, the arteries, capillaries, or veins. The heart itself, through degenerative changes in its muscle, as in fatty degeneration of the organ, may propel the blood with insufficient

[1] Cabot, *Boston Med. and Surg. Journ.*, March 22, 1894.

force. The presence of effusions in the pericardium may lessen the force of the heart's contraction. If an obstruction due to disease of the valves of the heart causes a narrowing of any of the orifices of the heart, the blood-pressure in the arteries falls, hence circulation is less active. If, as the consequence of disease of the valves of the heart, these structures do not entirely close their respective orifices, there is a backward flow of blood into the corresponding heart-cavity and the pressure in the vessels is lessened. Owing to degenerations of the walls of the vessels, mainly the arteries, they may lose their elasticity, and hence this element of equalizing blood-pressure fails and local circulation becomes disordered. The presence of a thrombus or embolus may mechanically impede the flow of blood to or from a part.

In any of these cases the tissues of the body suffer a disturbance of their blood-supply and are in danger of the hyponutritional changes described in Chapter IV. These changes in the vascular mechanism frequently occur at a time of life when the vitality of tissues is on the wane, so that degenerative changes are common. The changes in the vessels may be localized, in which case the nutritional effects are in correspondence.

Hyperæmia.—The term hyperæmia has reference to a localized condition. It means an increase in the activity of the circulation of a part. There are, however, conditions in which the entire vascular system appears to be overfull; the patient is said to be plethoric. Individuals who present this appearance are of two classes: those in which the vessels appear to be overfull and the circulation very active, or sthenic plethora, and those in which overfulness of vessels is associated with a sluggish circulation, or asthenic plethora. In sthenic plethora the general vital processes are active, and inflammations are quickly lighted up and run an active, but not dangerous course; the condition exhibits the phenomena of overfull arteries. On the contrary, in asthenic plethora, while the vessels are overfull, the circulation is sluggish and vital processes appear to be in correspondence. It is these two types which probably gave origin to the names of two of the basal temperaments, the sanguineous and lymphatic. Both conditions certainly exercise a governing influence in pathogenesis in the classes of clinical histories, prognosis, and treatment of diseases to which such persons fall victims.

The Pulse.—These and other conditions of the circulatory apparatus, are noted by studying the pulse—*i. e.*, the force, frequency, and regularity with which the blood is driven through the vessels. A knowledge of the general condition of the circulatory apparatus is gained by noting, first, the condition of the heart-beat. In a normal condition of the circulatory apparatus the heart-pulsations number from 70 to 80 a minute, accompanied by two distinct sounds with an interval between them, and

the sensation, when a superficial artery is pressed upon, of a quickly rising and quickly subsiding wave and with a moderate degree of tension, the artery filling like an elastic tube. Any variation in any of these particulars indicates necessarily a disturbance in the vascular mechanism. If the heart-sound is feeble, it indicates lack of power, the pulse-wave, on measurement with a sphygmograph, showing a short wave. If the contraction be slow, below 70, it is noted in the pulse; if both slow and weak, the pulse lacks tension, is compressible. If instead of the normal heart-sound a murmur or murmurs are heard, disturbance of the valves of the heart is indicated, and the tension of the arteries is below normal, the pulse-wave is disordered. In case of narrowing of the entrance of the aorta (aortic stenosis) the wave rises quickly and falls suddenly. In case the mitral valve fails to completely separate the auricle and ventricle in contraction, the wave is small and irregular. If the heart-sounds are pronounced, it indicates that a full volume of blood is being driven into the aorta.

Turning from an examination of the heart to a study of the arteries themselves, valuable indications as to the condition of the circulation are obtainable. It is to be recalled that the arteries are kept in a state of moderate contraction through the influence of the vasomotor nerves, probably by a balance between the influence of vasoconstrictor and vasodilator nerves. This point is, however, not well made out; it is not clearly determined whether dilatation occurs as the result of stimulation of one class of nerves, and contraction to stimulation of another class, or whether contraction is due to a set of nerves which, when dilatation occurs, are inactive. The latter hypothesis would imply that when vasomotor paralysis occurs the vessels are passively dilated to more than their normal size and are inelastic, this agreeing with observed clinical phenomena. The arteries are normally in a state of tension, so that when the blood-column contained in them is pressed upon by a mass of blood ejected from the left ventricle the column is impelled forward, which, meeting the resistance of the blood in the smaller vessels beyond the arterial trunk, causes an elastic distention of the artery.

The tension of the artery may be raised or lowered, according as the vessel is in more than normal or less than normal contraction. When the artery is in extreme contraction owing to overstimulation of the vasoconstrictor nerves, or from irritation of the vasomotor centre, the pulse is found to be small and hard; hard because of the tenseness of the arterial wall, and small for the same reason—the blood-column is unable to dilate the tense vessels.[1] This is the condition found in angina pectoris (neuralgia of the heart), in which the heart labors to overcome the spasmodic contraction and may suffer paralysis in its efforts. The

[1] Hare, *Practical Diagnosis*, 1896.

administration of the nitrites, amyl nitrite or trinitrin, paralyzes the nerve-centre, dilatation of the vessels occurs, and the heart is relieved.[1] Changes in the arterial wall due to disease of the coats may bring about a condition of heightened tension.

The conditions of the pulse are referred to as frequent or infrequent, corresponding with the heart-beat; as regular or irregular, corresponding with the heart-beat. The pulse may be full or small; full, relating to the volume and the extent of arterial expansion. Fulness is usually associated with strength of pulse, although not always. A small pulse is usually a weak pulse; but if the artery be much contracted, it may be both small and strong. The pulse may be soft or hard; hard in increased tension (see above); soft with diminished tension and diminished heart-power.[2] A hard, full, frequent pulse occurs in active inflammations. A hard pulse, full or small, bounding or not, if unconnected with acute symptoms, leads to suspicion of cardiac disease or an affection of the artery itself. A very frequent pulse, but feeble and compressible, is the pulse of marked debility, of prostration, of collapse.

It is by these several signs that the mode of distribution of blood throughout the body is gauged, although not positively determined, for disturbances in the circulation of a part may occur, and only be determined by a symptomatology referable to the part.

In the beginning of this chapter it was stated that there are two recognized types of hyperæmia: one in which the distention of vessels was upon the arterial, the other in which it is upon the venous side of the circulation. The two types differ as to causes, phenomena, and effects, and as to the indicated treatment for each.

Arterial Hyperæmia.

Arterial or active hyperæmia is an increase in the amount of blood in the dilated arteries of a part.

Causes.—The direct cause of hyperæmia of a part is a lessened arterial resistance; the tension of the arterial walls is lessened by a stimulation of the vasodilator nerves or by a sedation of the vasoconstrictor nerves, the former being the more probable explanation. It expresses the reaction which occurs as the consequence of the presence of an irritant. Alternate rise and fall in arterial tension occur in health; it is only when the condition is prolonged that it becomes pathological. If the sensory nerves of a part are stimulated or irritated, hyperæmia of the irritated parts occurs as a reflex. If the surface of the body be irritated at certain points, as by the application of heat, it appears that in some cases hyperæmia may be induced in deeper-seated organs as a reflex.[3]

[1] Brunton's *Pharmacology.* [2] DaCosta, *Medical Diagnosis.*

[3] Green's *Pathology and Morbid Anatomy.*

Hyperæmia may be compensatory, as when, through the removal of one of a pair of organs having the same function, an increased blood-supply is present, and increased work is performed by the remaining organ.

Symptoms.—The symptoms accompanying hyperæmia of a part are such as would be directly surmised as soon as the condition was defined. There are increased redness, an elevation of temperature, and more or less throbbing, and in some cases some degree of throbbing pain.[1]

Pathology.—The arteries are dilated: there is an increased flow of blood through them and also to them through their own nutritive arteries; the pressure in the veins rises, but exudation does not appear to increase in all cases, as there is no increase of lymph-pressure, although in marked cases œdema may occur. Two types of arterial hyperæmia appear to exist: one due to excitation of the vasodilator fibres, a neurotonic congestion; the other, a neuroparalytic congestion, caused by paralysis or sedation of the vasoconstrictor fibres (Recklinghausen).

Results of Active Hyperæmia.—The nutrition of the part is increased; there is in the less pronounced cases an increase in the functional activity of the part; secretion is increased; the vital activities are elevated. In more pronounced cases, where the symptoms of redness, heat, swelling, and throbbing pain occur, the function of the part is disturbed. Many of the cases formerly included among the mild inflammations are now placed under the head of active hyperæmia.

The character and composition of the exudates differ widely in the two conditions:

Hyperæmic Exudates.	*Inflammatory Exudates.*
Poor in albumin.	Rich in albumin.
Rarely coagulate in tissue.	Usually coagulate in tissue.
Contain few cells.	Contain numerous cells.
Low specific gravity.	High specific gravity.
Contain no peptone.	Contain peptone.[2]

The lesser forms of hyperæmia if continued may lead to hypertrophy of the affected organ and of the vessels. In the more marked forms the changes induced approach the degenerations.

Treatment.—The principle of treatment is to remove the cause and procure surgical rest. The source of the irritation is to be sought out and removed when possible; as a rule, the symptoms then promptly subside. It may be that the conditions existing require treatment irrespective of the cause, which may not be determined or be absent, the vessels being dilated as the effect of a previously acting cause. The principle of treatment is the reduction of the dilated vessels. This is attempted at times through the use of drugs; for example, the adminis-

[1] Warren, *Surgical Pathology and Therapeutics.* [2] Park's *Surgery*, vol. i., p. 25.

tration of ergot, which, by stimulating the vasoconstrictor system, lessens the calibre of the dilated vessels. The antagonist of ergot, aconite, has also been used. By paralyzing the vasoconstrictor system and quieting the usually overacting heart-muscle it lessens the amount of blood flowing to a part in a given time.

The usual method of inducing contraction is by local applications of dry cold, which cause contraction of the arteries.

The principle of derivation is also employed: by local bloodletting, by incision, wet cups, or leeches applied beyond the margins of the hyperæmic area, the engorged vessels are unloaded. Frequently the administration of a diaphoretic or diuretic, by diverting the blood-current, causes a lessened flow to the affected area. The administration of a saline cathartic lessens the fluid volume of the blood, and, conjoined with local measures, reduces the engorged vessels.

What is known as counterirritation is a common means of treatment. An irritant, such as a mustard-plaster or a blister, applied at a distance from the affected part, induces a flow of blood to the point of application and lessens the amount of blood in other parts. The volume of the blood being in definite amount, if an excess exist in any part, a deficiency will be found in other parts.

Sedative astringents (the liquor plumbi subacetatis) are used to contract dilated vessels. Other astringents (see Pharmacology) induce dilatation of vessels, and so are contraindicated.

Perhaps the most effective measure is local bloodletting, and in the more continued cases the repeated application of cold.

Venous Hyperæmia.

By venous hyperæmia is meant an excess of blood in the dilated veins of a part.

Causes.—Its causes are mechanical interference with the return of the blood to the heart.

Symptoms.—The symptoms of this condition are blueness (instead of redness), a lessened temperature, and swelling.

Pathology and Morbid Anatomy.—The veins are dilated, the current is slowed, and intravenous pressure is increased, in consequence of which watery exudations occur in the parts about them. The deficiency of arterial and the excess of venous blood, with interference with its return to the heart, are followed by deficient supply of oxygen, a lessened food-supply, and the retention of waste-products: the effects are in correspondence, vital processes are lessened, secretion is diminished, there is less oxidation, and hence less heat is produced and less work is done. The functional activity of the parts suffers, and degenerations, atrophy, or necrosis may occur.

Treatment.—The principle of treatment is the removal of the mechanical obstruction to the return of the blood and mechanical support of the engorged vessels. This latter is accomplished by means of elastic bandages, and in situations in which these cannot be used, astringents may be employed. The part is elevated when possible, to aid in the return of the blood to the heart.

The succeeding type of tissue and vascular reaction toward an irritant—inflammation—must be treated as a process *sui generis*, although exhibiting many features of pronounced arterial hyperæmia.

Inflammation.

There is no condition described in pathology which has received such a variety of definitions as that of inflammation. A general consensus of opinion places inflammation in the category of the conservative processes, viewing it as essentially Nature's means of ridding the tissues of an intruding substance—an irritant.

As a general description, there is perhaps no better definition than that of Sanderson:[1] "Inflammation is the succession of changes which occurs in a living tissue as the result of some kind of injury, provided that this injury be insufficient to immediately destroy its vitality."

A definition which expresses the pathological features found in this condition better than any other is that of Ziegler:[2] "It is essentially a local tissue-degeneration combined with pathological exudations from the bloodvessels, followed sooner or later by tissue-proliferation, leading to regeneration or hypertrophy."

It has been suggested[3] that the term inflammation be dropped from use, as it includes so many factors which are variable in occurrence as to give the word but indefinite meaning.

Causes.—The causes are injuries of any description which induce a higher degree of irritation than those producing active hyperæmia. The sources of these irritations or injury are many and varied. They may be included under the heads of mechanical violence, the action of physical forces, the action of chemical substances, and the action of parasites and their products. Any one or more of these influences acting upon a vital part may induce the inflammatory process.

The causes may be extrinsic or intrinsic; while most of them act from the exterior of the body, substances formed in the body, such as uric acid, are sufficient irritants at times.

Inflammations which are caused by the action of physical or chemical agencies are usually termed simple; those arising through the in-

[1] Holmes' *System of Surgery*, vol. i. [2] *General Pathology*, 1895.
[3] Thoma, *General Pathology*, vol. i.

fluence of vegetable parasites or their products are named infective inflammations.

Pathology.—Inflammation may be fitly regarded as one stage of a series of nutritive disorders which begins in stimulation and ends in necrosis. It is a profound disturbance of the nutritive functions of a part. The pathology of inflammation is studied by inducing the condition in transparent and vascular membranes of animals, which may be fixed to the stage of the microscope.

The first effect of the application of a sufficient irritant to a part is the quick contraction of the arteries, followed immediately by a dilatation of arteries and veins, the velocity of the current increasing; the blood-pressure is at this stage apparently not marked, for there is but

FIG. 38.

Inflamed human omentum. The phenomena of inflammation are seen in the veins and capillaries, the condition being normal at the artery (*c*), where *b* represents endothelium covering the trabecula (*a*). In the vein (*d*) there are many white corpuscles along the wall: some of these are emigrating (*e*); *f*, desquamated endothelium; *g*, extravasated red corpuscles. (Ziegler.)

little dilatation of the capillaries. Up to this point there is nothing observed which could not be explained by a paralysis of the vasoconstrictor or stimulation of the vasodilator nerves; the process of inflammation proper begins when the blood-stream in the emergent veins is seen to be retarded and an exudation is poured out of the small veins and capillaries; with the slowing of the current the increased blood-pressure causes a marked dilatation of the capillaries. An evident change may now be noted in the condition of the walls of the vessels.[1] The leuco-

[1] Cohnheim and Samuel.

cytes, which before the inception of the condition were few and scattered, are seen from the beginning of this process of retardation to cling and mass in numbers along the walls of the smallest veins, the large mononuclear and the multinuclear forms particularly. Instead of the usual exudate of lymph, the vessels of the area now pour out an abundant exudate rich in albumin, and soon the white corpuscles may be seen making their way through stomata of the veins to the intercellular spaces (Fig. 38). The area about the bloodvessels is soon filled with a mass of mobile cells. The origin of all these cells is a mooted point. It was taught by Virchow that they arose from the multiplication of the connective-tissue corpuscles of the part; and later, their origin was stated by Cohnheim to be solely from the leucocytes which had made their escape from the vessels. It is the belief at present that they arise mainly from the leucocytes at this stage of the inflammatory process.

The diapedesis is explained by Metchnikoff as Nature's defence against an intruding and injurious body. When such a body, or when a source of marked irritation is present, the leucocytes throng to the point of irritation, emerge from the vessels, and attack the intruder, exercising their phagocytic activity against it. If the intruder is of smaller size than the leucocytes (as bacteria), it is enclosed by the latter, killed, and digested. Large bodies are attacked and surrounded *en masse* by leucocytes. If the phagocytic cells conquer, the acute symptoms subside and a series of changes occur which lead to a restoration of health. If the bacteria or other intruders prevail, the phagocytes succumb, die, and form what are called pus-corpuscles.

The blood in the capillaries comes to a standstill, a condition of stasis. Coagulation of the blood, however, does not occur, as a rule; for if the blood-flow be re-established, the separate red corpuscles are seen, one by one, to roll away from the general mass until all are in movement and stasis ceases (Thoma). The affected area becomes filled with a mass of indifferent cells and remnants of broken-down tissue, for it appears that the nutritive balance of the tissue-elements of the part is entirely lost, and that profound degenerative changes occur in them; it is believed, therefore, that the source of the cells at the height of the inflammatory process is from the leucocytes alone. The albuminous exudate, in the presence of injured leucocytes, is in fit condition to coagulate, and coagulation of the effusions occurs. The area of inflammation now represents virtually a mass of embryonic tissue containing foreign substances. The inflammation may take one of two courses: the inflammation may abate, and a restoration of health and regeneration of tissue in the parts take place; or the cells of the exudation, together with more or less of the tissue, may die. In the former case the process is called resolution; in the latter, suppuration.

It is interesting, in this connection, to review the beliefs as to the causative agents in inflammation. It has been demonstrated quite clearly that the process of suppuration occurs only in the presence of bacteria or their waste-products. It is held by one school of pathologists that true inflammation also occurs only in consequence of the presence of similar causes, the inference being drawn mainly from Metchnikoff's observations. Another school hold that inflammations are of two types —simple and infective. If suppuration occurs only in the presence of bacteria or their products, then pus-formation cannot be a result of what are termed simple inflammations. We are compelled, therefore, either to believe that the evidence as to the invariable association of pus and bacteria is insufficient and faulty, or else hold that all inflammations are bacterial. The evidence as it exists divides inflammations into two classes: first, those certainly infective; secondly, those not certainly infective.

Symptoms.—The classical symptoms of inflammation are *rubor, tumor, dolor, calor, et functio laesa*, or redness, swelling, pain, heat, with disturbance of function. The general symptoms are usually an increased heart-action, some elevation of the temperature of the body, and disturbances in secretions. The redness is due to an increased amount of blood in the part, the color being deeper in the centre of the inflammatory area. The swelling is due to the exudation; the hardness of the swelling being due to coagulation of the effusion. The pain is the result of the pressure of the effusion upon sensory nerve-terminals; it is frequently throbbing, in correspondence with the heart-beat. The heat is owing to the greater amount of arterial blood carried to the part; experiments to show that there is an increased generation of heat in an inflamed part have resulted negatively.[1] The function of the part is disturbed from mechanical and vital reasons; the presence of the exudation itself tends to disturb functional activity, but there is, beyond doubt, in inflammation a profound disturbance of the nutritive balance in the tissues. Any one or more of these symptoms, except disturbance of function, which is a truly cardinal symptom, may be absent in some anatomical situations.

Seat of Inflammation.—Virchow divided inflammations into parenchymatous and interstitial, according to the portion of the organ attacked. Parenchymatous, those in which the functional cells of a part were affected; interstitial, those in which the connective tissue was affected. True inflammation occurs only in the perivascular connective tissue, so that it is always interstitial. It has been suggested that the term parenchymatous inflammation be abandoned, but there is no other term at present which expresses the condition—a profound disturbance

[1] Warren's *Surgical Pathology and Therapeutics*, 1895.

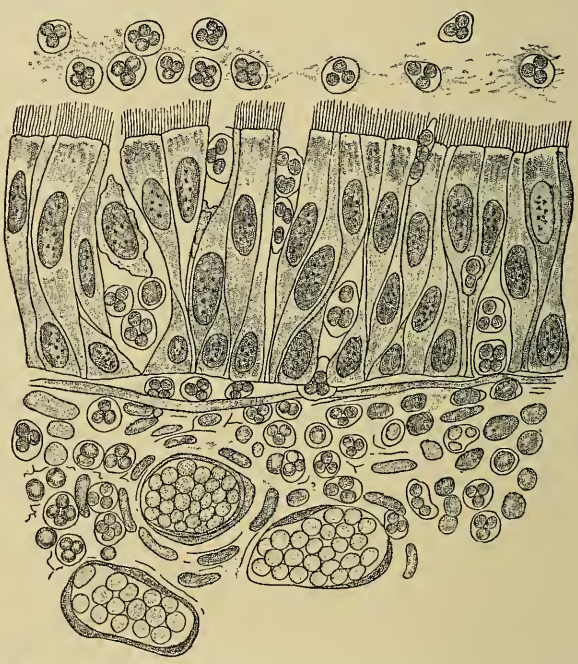
of the vascular supply underlying these cells, accompanied by a granular degeneration of the cells and their death.

When the effusion contains a small amount of albumin and corpuscles the condition is called a serous inflammation.

In effusions largely corpuscular coagulation occurs, and a fibrinous inflammation is spoken of.

If the effusions and corpuscles make their appearance upon the surface of a mucous membrane, and there is more or less swelling and desquamation of the epithelial cells, the condition is termed a catarrhal inflammation (Fig. 39).

FIG. 39.



Acute bronchial catarrh: passage of leucocytes through the epithelium of the bronchus between the ciliated cells. × 700. (Thoma.)

Treatment.—The cardinal principle in the treatment of inflammation is the removal of its cause. As inflammation is Nature's means of ridding herself of an irritant, the removal of the irritant by the operator is the most efficient aid in the process. As the majority of inflammations are certainly due to the action of bacteria, the removal of bacteria and their products is the principal aim of treatment (see Chapter VI.).

Aside from the consideration of removing the cause, the treatment of inflammation is directed to modifying or removing the several conditions which give rise to the symptoms. The tissues are overloaded with

exudation which the lymphatic vessels cannot remove; the bloodvessels are distended by an increased amount of blood, whose exit is blocked, but which is receiving additions with each pulse-beat, increasing the pain, swelling and heat. The indication is clear—relieve the stagnation. The direct causative factor of the stagnation is the vascular supply; if the blood can be made to flow freely along the clogged veins, the flow through the capillary area will also be re-established. If less blood is carried to the part, there will be no additional stagnation. So long as the vascular congestion persists there is no possibility of removal of the effusion by the lymphatics.

Nanerede found, on dividing a vein upon the distal side of an area of inflammation, that after a brief period the flow of blood was established through the inflamed area. Local bloodletting by leeches (Gensmer) produced even more marked effects. Drugs which stimulate the vasoconstrictors (ergot), and those which paralyze the constrictors (aconite), lessen the blood-pressure in the inflamed area, so that if administered in the early stages of inflammation they may modify its severity. If, on the contrary, they are administered after stasis occurs, they increase the stasis—ergot actively and aconite passively. If the flow of blood through the inflamed area is re-established by local bloodletting, after the period of stasis, then the arterial sedatives are distinctly useful in lessening the flow of blood to the part.[1]

When, owing to vascular engorgement, throbbing pain is a prominent symptom, applications of cold are useful in lessening the calibre of vessels and in relieving pain. But if there be firm exudation and marked stasis, cold is a detriment. Heat then gives relief through inducing a more free flow of blood in the collateral circulation. Very hot applications act as do cold applications, by causing contraction of vessels, and may be used to abort an inflammation.

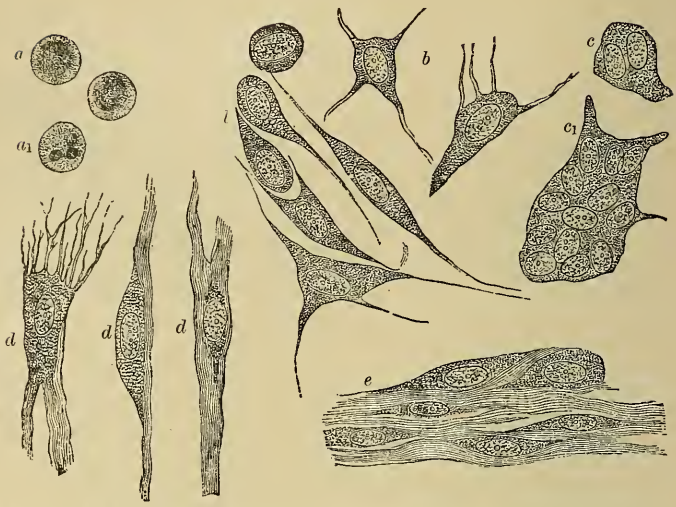
General sedatives are at times demanded for the relief of pain. Morphia, used in small and continued doses, not only relieves pain, but causes a contraction of small vessels.

Regeneration of Tissues.

When the inflammatory action terminates without the formation of pus a series of changes is instituted which normally results in a reorganization of the area of degeneration. This is termed the process of repair. The pathological exudations cease, the vessels resume their normal tone, tissue which has died as the result of the inflammation is cast out or is absorbed (eaten by the phagocytes) and removed, and the exudate is absorbed by the lymphatics. The site of inflammation is now filled with a mass of indifferent embryonic corpuscles, consisting in

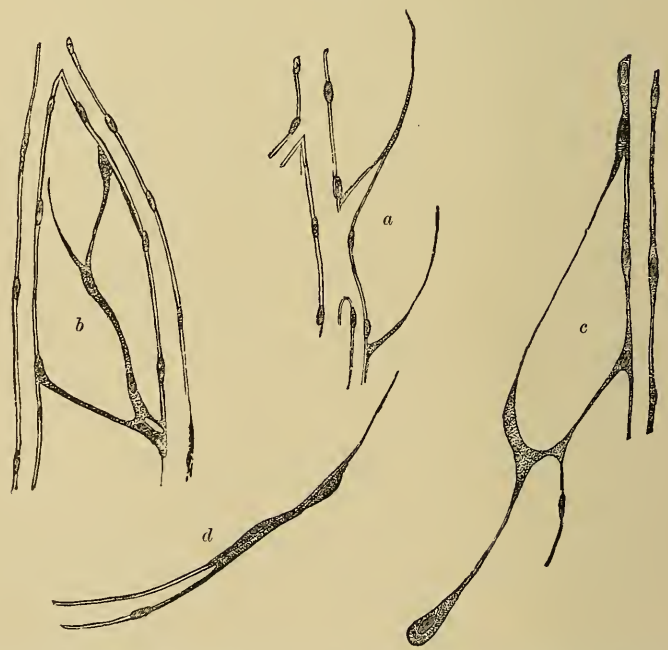
[1] Warren, *Ibid.*

FIG. 40.



Isolated cells from a granulating wound: *a*, uninuclear leucocyte; a_1, multinuclear leucocyte; *b*, different shapes of uninuclear formative cells; *c*, double nucleated formative cells; c_1, multinucleated formative cells; *d*, formative cells in the process of tissue-formation; *e*, completed connective tissue. Picrocarmine preparation. × 500. (Ziegler.)

FIG. 41.

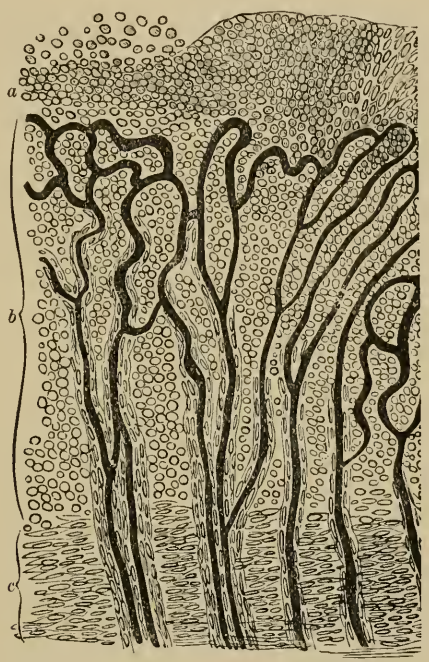


Development of a bloodvessel by formation of offshoots, from preparations which were taken from a formation of inflammatory granulations: *a*, *b*, *c*, *d*, different forms of offshoots—some solid (*b*, *c*), some becoming hollow (*a*, *b*, *d*), some simple (*a*, *d*), some branching (*b*, *c*), some without nuclei (*a*, *d*), some with nuclei (*b*, *c*). Formative cells have applied themselves to the outside of the offshoots. (Ziegler.)

part, no doubt, of embryonic cells of the several tissues destroyed by the inflammatory process, among which are numbers of leucocytes (Fig. 40). It is out of this embryonic tissue that a reproduction of the several tissues takes place. It is to be remembered that while any of the several connective tissues—the tissues of mesoblastic origin—may be reproduced from this embryonic tissue, epithelial tissue is never reproduced save from epithelium.

The embryonic tissue soon acquires a new blood-supply (Fig. 41). In the endothelial cells of the walls of the capillaries about the part division of the cell-nucleus takes place, and by the reproductive process

FIG. 42.



A granulating surface: *a*, layer of pus; *b*, granulation-tissue with loops of bloodvessels; *c*, commencing development of the granulation-tissue into a fibrillated structure. × 200. Diagrammatic. (Rindfleisch.)

a bud grows from the capillary wall; by repeated reproductions solid columns of cell-substance are formed which, joining columns from other capillaries, form loops, the centre of the columns becomes hollowed out, forming tubes which transmit blood—*i. e.*, new capillaries are formed. The mass of embryonic tissue becomes thus permeated by a network of new capillaries (Fig. 42). The tissue in its present state is known as granulation-tissue. The cells of granulation-tissue are partly hypertrophied tissue-cells and partly mono- and polynucleated leucocytes.[1]

[1] Ziegler's *General Pathology*.

When the cells have organized and are transformed into connective-tissue forms and arrangement, contraction takes place, and most of the new bloodvessels are obliterated; the new tissue becomes pale—it is a cicatrix. The indifferent embryonic cells may have the function of forming any of the connective tissues. If cartilage is to be formed, chondrification takes place about the specialized cells. If bone is to be formed, each cell becomes an islet around which calcification proceeds. If epithelium have been lost, the epithelium existing undergoes proliferation, growing inward from the sides of the area denuded of epithelial covering, until a new epithelial surface is formed.

CHAPTER VI.

INFECTIVE INFLAMMATIONS: SUPPURATION, ABSCESS, FEVERS, SEPTICÆMIA, AND PYÆMIA.

An infective inflammation may be defined as a condition in which the phenomena described under the head of inflammation are caused by or characterized by the presence and development of pathogenic bacteria. In the light of Metchnikoff's studies, many pathologists maintain that inflammation is always an infective process; that it is in consequence of the presence of bacteria that the phenomena of inflammation occur. This view is not entirely subscribed to, particularly by German pathologists; but the belief is daily gaining ground among the pathologists of other nationalities that Metchnikoff's theory, even though not meeting all conditions, furnishes the most satisfactory explanation of the etiology of inflammation ever presented. This theory is, in substance, "that the process of inflammation is one of the factors in organic evolution. Beginning with the amœba, this simple organism, the analogue of the white blood-corpuscles has the power of englobing and digesting solid matters, such as bacteria, with which it is brought in contact." In animals progressively higher in the zoölogical scale, wandering cells of the body, similar to the white blood-corpuscles, exhibit this property, until in the mammalia, including man, Metchnikoff holds that the same cells have as a distinctive function the attacking and removing of foreign substances, pathogenic organisms included, which gain access to the body. As noted in discussing the subject of bacteria (see Chemotaxis), special properties of the invading substance may determine whether it is to be attacked and removed by the white blood-corpuscles, or whether it is avoided by these same cells. He sums up his investigations, made upon all classes of animals, and after reviewing the experiments of Virchow, Samuel, and Cohnheim, with the dictum that "*The essential and primary element in typical inflammation consists in a reaction of the phagocytes against a harmful agent.*" [1]

Metchnikoff and his followers maintain that the changes which occur in the absence of bacteria are not characterized by the flocking and diapedesis of leucocytes; that this latter phenomenon is, in fact, the distinguishing feature of inflammation; and that conditions following injuries of other kinds belong to the hyperæmias.

[1] Metchnikoff, *Comparative Pathology of Inflammation*, 1893.

Leaving open the question, whether true inflammation can occur in the absence of bacteria, it is certain that a vast majority of inflammations are due to or are characterized by the development of pathogenic organisms at some point—the area of infection. That is, when pathogenic bacteria gain entrance to the body and find a suitable soil, they undergo multiplication, they cause degeneration in the tissues; inflammation with death of more or less tissue, and, after a period, regenerative changes occur. During the period of their growth substances are formed which, being taken into the circulation, act as poisons (see Fever, Septicæmia, and Pyæmia). For example, in typhoid fever a specific bacillus gaining access to the glands of Peyer's patches, *under favorable conditions* multiplies and causes degeneration, inflammation, and cellular necrosis of the part, forming ulcerous patches. During the period of multiplication toxic substances are formed which, when absorbed and carried by the circulatory fluids throughout the body, cause the train of symptoms peculiar to typhoid fever. The nature of the local changes induced and the general symptoms accompanying infection depend upon the nature of the infecting organism. The effects of pathogenic organisms as a class are, however, those above noted: local tissue-degeneration, inflammation, cellular necrosis, with symptoms of general poisoning; at a later period regenerative changes occur which heal the injured part. This naturally divides the study of infection into two heads: first, local effects; secondly, general symptoms.

Suppuration.

By far the most common and universal causes of infective inflammations are the pyogenic cocci (p. 45), organisms which, gaining access to the interior of the body, cause the formation of pus. According to their mode of grouping, these cocci are divided into two classes: first, the staphylococci; secondly, the streptococci. The staphylococci are subdivided according to the color of their colonies in a culture-medium: the staphylococci producing orange-colored colonies are called staphylococci pyogenes aureus; those forming whitish colonies, the staphylococci pyogenes albus; those causing lemon-colored growths, the staphylococci pyogenes citreus; those giving green colonies, the staphylococci pyogenes viridis. The staphylococcus pyogenes aureus appears to be everywhere.

Bacteria may gain entrance to the body through wounds of the skin or of mucous membranes, or through any break or abrasion of these surfaces. They enter nearly every wound made, unless special precautions are taken to exclude them; they are inhaled, and may be taken into the body from the respiratory tract; they may be taken up from the alimentary tract.

PLATE II.

Abscess in Kidney of Rabbit after Intravenous Injection into an Ear-vein of Culture of Pyogenic Cocci. Dense mass of cocci surrounded by area of coagulation necrosis due to their toxic activity. Outside this a zone of phagocytes.

It is of extreme importance to remember that a condition of perfect health in cells, tissues, and the body, is a safeguard against the attacks of any organisms which may gain entrance to the body ; and, *vice versa*, a susceptibility to their action is produced in the body, in its tissues, or in its cells, by a condition of debility of a part. This is notably true of hyperæmia ; a part which is suffering from the degree of irritation which causes hyperæmic debility, will fall a ready victim to the action of organisms which would probably not affect it in a condition of health. To take a simple example : the mouth of a sebaceous gland of the skin becoming occluded, there is no escape for the secretion, which accumulates and changes in character ; owing to the mechanical irritation of accumulated products, secretion is at first increased, until the acini and duct of the gland become much dilated ; the part becomes like a foreign body, is a source of irritation and active hyperæmia results. Due to this cause, and, no doubt, to fermentative changes which occur in the accumulated secretion, a condition of lessened resistance is produced. The ever-present staphylococcus pyogenes aureus gains access to the gland, multiplies, and causes the changes characteristic of an infective inflammation. The phenomena of inflammation occur—heat, redness, pain, and swelling ; there is a diapedesis of white blood-corpuscles with a fibrinous effusion, which in the presence of injured leucocytes coagulates. In the struggle between the invading bacteria and the leucocytes myriads of the latter have succumbed—have died ; the organisms secreting or excreting a substance capable of changing the (coagulated) albuminous effusion into liquid peptone, there results a fluid holding in suspension the leucocytes which have died, and the detritus of the tissue destroyed in the struggle ; the tissue-destruction proceeds in all directions, advancing most readily in the lines of least resistance, until the accumulated fluid, with the cells and detritus, are discharged. Occurring in a very small area, the process describes the origin, course, and termination of a pimple ; if a larger territory is involved, a boil ; in both cases the condition is one of abscess. The fluid discharged from the abscess is called pus. It consists of inflammatory effusion, now fluid, having been transformed into peptone, the dead leucocytes, dead bacteria, and the remnants of broken-down tissue. This is the essential process of suppuration in any part : the entrance of pyogenic organisms into the tissues, the exciting of inflammation at some point of lessened resistance—a *locus minoris resistentiæ*, the death of tissues and leucocytes, the peptonizing of inflammatory effusions and breaking down of dead tissue, and the escape of the fluid thus constituted (Plate II.).

It does not always happen that the pus finds escape, either naturally or through surgical aid ; the patient may die before this occurs (see

Septicæmia), or the tissues beyond the seat of pus-formation, those in which the irritation does not exceed constructive hyperæmia, may form a boundary-wall which the organisms fail to break down and thus die—starved out; the abscess-contents undergo changes, a cheesy or, in some cases, a calcareous mass marking the site (see Degenerations).

While the staphylococci cause, as a rule, the circumscribed destruction of tissue described, the streptococci, as, for example, the streptococcus of erysipelas, tend to multiply laterally, following the tortuosites of the connective tissue of the skin. They give rise to a progressively spreading violent inflammation, but do not cause a marked peptonizing of effusions and dead tissue—*i. e.*, but little pus is formed.

Cause.—The cause of suppuration is the development in tissues of pyogenic organisms.

Symptoms.—The symptoms of suppuration are both general and local. The local symptoms are, first, those of inflammation. At the height of the inflammation the apex of the swelling, hitherto of marked firmness, gives a feeling of lessened resistance, and later acquires a boggy softness. If the swelling be of marked size, distinct fluctuation may be felt, showing the presence of fluid; the apex of the swelling bursts, giving vent to the abscess-contents, when the inflammatory symptoms rapidly subside, the tissues lose their hardness, and usually regenerative changes occur, causing the obliteration of the abscess-cavity and tract; or it may be that pus-formation continues after the subsidence of the inflammatory symptoms. The area of tissue-loss is called the abscess-cavity; the pathway leading from the cavity to the point of discharge is called the fistula.

General Symptoms.—The general symptoms of suppuration are caused by the absorption of the products of bacterial growth. These symptoms, if the suppuration be extensive, may be ushered in with a chill; there may be fever as high as 104° F., a full, bounding pulse, and all the accompaniments of fever. In some cases of delayed evacuation of the abscess there may be evidence of profound poisoning.

Prognosis.—As a rule, abscesses tend to spontaneous recovery after evacuation, and in case a persistent discharge remains it is to be suspected that some portions of dead tissue have not been discharged, and that in minute crypts or crevices bacteria still develop. The occurrence of rigors (chills) and high fever is a danger-signal. A fluttering, weak pulse and clammy extremities following upon the primary fever are evil omens.

Treatment.—When it is considered that the disease symptoms and dangers attendant upon the process of suppuration are due, in the first place, to the pyogenic organisms, and that the presence of pus and dead tissue is a source of debility and a detriment to the process of regenera-

tion, it is at once clear that the therapeutic indication is to rid the body of all of these substances; that is, the evacuation of the pus is always the measure to be adopted.

As soon as it is determined that pus is present in any accessible situation whatever, it should be given vent. If it be an abscess pointing, as in a boil or over the root of a tooth, a sharp-curved bistoury should be passed through the top of the swelling and by a drawing motion a linear cut made, giving free exit to the pus. Even though the pus be not nearing a natural vent, if it is enclosed in the cancellated tissue of bone (osteomyelitis), or, as in alveolo-dental abscess, confined under the maxillary periosteum (see chapter on Alveolar Abscess), or be under the periosteum of the finger or elsewhere, an opening should be freely made into the infected area. As a rule, healing is spontaneous after the opening of an abscess, the discharge lessening gradually, and the cavity and exit being filled with granulation-tissue. In case the abscess-walls contain crypts in which bacteria may multiply, it may be necessary to use antiseptic washes to reach and destroy them. Hydrogen dioxid, in a 15 volume or 3 per cent. aqueous solution, is used to syringe out the cavity. Should the opening of the cavity be large, and there be danger of reinfection, it is advisable to pack the cavity with gauze impregnated with iodoform or aristol, or with the more recent iodin preparation, nosophen. Any dead tissue in the abscess must be mechanically removed, as regeneration will be delayed or prevented so long as the necrosed parts are suffered to remain.

ULCERATION.

The development of the pyogenic organisms upon a free surface causes tissue-degeneration and death, as described under abscess; in fact, an abscess is a confined ulcer.

Numerous forms of pathogenic organisms are capable of causing tissue-degeneration and death of a mucous or skin surface. If infection takes place through a hair-follicle, or if organisms develop upon an abrasion, or in the epithelium, in conditions of general or local debility, the epithelium is destroyed over an area, and in the subepithelial tissues the organisms multiply and cause tissue-loss. If the organisms be pyogenic—and ulcerous surfaces are usually infected by these bodies—pus is formed. Under some conditions, as in debilitated and neglected children, the ulcerous process may spread rapidly, as of the cheek in cancrum oris; or when specific bacilli, which excite much swelling and quick death of the tissues of the cheek proliferate, causing the condition called noma.

Treatment.—The general treatment of ulcers is to destroy the infecting organisms by antiseptic washes, such as a 1 : 2000 solution of

mercuric chlorid in hydrogen dioxid, and subsequently washing the surface of the ulcer with some powerful antiseptic which destroys the superficial tissues and the bacteria in them; for example, concentrated carbolic acid. The exposed surface is next covered with an antiseptic powder—iodoform, aristol, or nosophen (see section on Pharmacology)—and then the surface is to be protected from the access of organisms until the regenerative process has healed the breach. It is to be remembered, in this connection, that the reproduction of epithelium is from the epithelial boundary of the ulcer, for epithelium cannot develop from any but epithelial tissue.

OSTEOMYELITIS.

There is one kind of suppuration of more than ordinary interest to the dental practitioner, as beyond doubt many cases of maxillary abscess should be classed under this head—osteomyelitis, an inflammation of the bone-marrow.

Causes.—In conditions of debility pyogenic organisms, notably the staphylococci, and occasionally streptococci or pneumococci, or the bacillus tuberculosis, and in special cases the typhoid bacillus,[1] gain access to the cancellated tissue of bone; they cause a thrombosis of the vessels about the area of infection, the bone-cells die, and suppuration ensues.

Pathology and Morbid Anatomy.—In consequence of the growth of the organisms the adjoining vessels become thrombosed, the cells of the bone die—undergo coagulation-necrosis,[2] and the tissues are rapidly broken down into pus. The process spreads until the bone is perforated and the pus finds exit beneath the periosteum, which it separates from the bone unless the exit be immediate, and necrosis of that portion of the cortical bone results. If the process approach the nutrient artery of the bone, thrombosis of that vessel occurs; and if it be a trunk of large size and of very limited anastomoses, as in the inferior dental artery, necrosis of the bone results. The case is one of abscess in peculiar anatomical situation, where the ordinary phenomena of inflammation can not be manifested.

Symptoms.—The general symptoms of this condition are those of septic infection, combined with local evidences of marked disorder. There is debility with sharp but ill-located pain, which may be followed by a chill, high fever, and a local inflammatory reaction of much severity; the fever assumes the adynamic type. The pain becomes localized in the affected bone, and the duration of the symptoms will depend primarily upon the time required for the escape of pus, naturally or through surgical aid. When the superficial layers of a bone

[1] Park's *Surgery*, vol. i. [2] *Ibid.*

are affected or the infection is subperiosteal, it constitutes the condition known as acute infectious periostitis.

Treatment.—The essential principle of treatment in all cases as soon as recognized is to gain access to the infected parts by incising and drilling instruments, drain the cavity, remove dead tissue, and sterilize the cavity.

Fever.

The term fever is applied to a condition the most prominent feature of which is an elevation of the bodily temperature above the normal, 37° C. To constitute a fever this rise in temperature must continue for some length of time.

Causes.—Fevers are commonly caused by the presence in the circulatory fluids of substances which act as poisons upon probably the nerve-centres controlling heat-production. As a rule, the offending substance is a poison generated in the body through the action of micro-organisms. The character and type of the fever are determined by the nature of the offending substances—*i. e.*, the variety of infection.

Classes.—Fevers are divided into periodic or continued, according as to whether there is a periodical fall of temperature and a subsequent rise, or whether the fever continues practically unabated from the beginning to the termination of a disease. Fevers are classed in severity according to the maximum temperature and again according to their duration. A temperature of 100.5° to 101.3° F. is called slightly febrile; 101.3° to 103° F., moderate fever; 103°–105° F., marked fever. A temperature above 106° is termed hyperpyrexia.

Symptoms.—The most characteristic symptom of fever is the elevation of temperature; accompanying this there is an increased frequency of the pulse. In acute inflammatory diseases the pulse is full and bounding, the eyes injected, the bowels constipated, and the urine scanty, containing an excess of urea. On standing, the urine throws down a brick-dust deposit (urates). In fevers of a lower type, or in many fevers which began as described, the high, bounding pulse is succeeded by a soft, quick pulse and evidences of great debility. In fevers in which the temperature runs high there is commonly evidence of intoxication, more or less delirium, and reflex muscular action. With a persistent temperature and a pulse becoming softer and more frequent, there is increasing debility.

Pathology and Morbid Anatomy.—In all cases of continued high temperature the fat of the body rapidly disappears and granular degeneration occurs in the muscles and viscera of the body. If the fever be long continued and of an adynamic type, this degeneration may become marked. Its occurrence in the muscles of the heart is common and is

an element of danger. There are an increase in the amount of carbon dioxid formed and exhaled from the body, and an increased amount of oxygen inhaled. This, with the increase of urea, the product of the oxidation of nitrogenous tissues (muscles, glands, etc.), indicates that the oxidation of the tissues is largely increased; hence the elevation of temperature. As repair does not equal waste in fevers, the nutritive processes being profoundly disturbed, the essential elements of the tissues suffer from the increased oxidation and undergo degenerative changes.

Prognosis.—The higher the temperature and the longer it continues the greater drain there is upon the vital forces. As a rule, a temperature of 106° F. persisting more than twenty-four hours presages death. If the vital forces flag and the heart-action becomes weakened, and if there be evidence of profound intoxication, such as twitching of tendons, low, muttering delirium, and a clammy surface, the outlook is bad. Favorable signs are falling temperature, a clear eye, tongue losing its coating, free action of the bowels, free perspiration, free action of the kidneys, and a good vascular tension.

Treatment.—In the light of present knowledge, efforts should first be made to discover the nature of the cause of the fever and to remove it, if possible. If not, attention should be directed to maintaining the vital forces until the body rids itself of the offending causes. As many fevers are self-limited in course and duration, this latter treatment becomes an important consideration. Temperature should be kept within safe limits by the administration of antipyretics, when the condition of the heart will permit their being used, and also by cool sponging or cool baths. The action of the heart should be sustained by the administration of concentrated nutriment, and by stimulants when necessary. The bowels must be kept open.

In any form of fever there is no therapeutic measure comparable with removal of the cause, provided this be discoverable, identified, and removable.

Septicæmia.

Septicæmia is a condition in which septic matter is present in the circulating fluids of the body, and causes manifestations of widespread disorder.

"The essence of septicæmia is a poisoning of the organism by toxins, toxalbumins, ferments, and other products of bacterial decomposition—*i. e.*, it is septic intoxication."[1]

Varieties.—Conditions of septicæmia may be classified, according to the severity of the symptoms, as mild septic intoxication, septic poisoning, and pyæmia. The nature and severity of the intoxication depend

[1] Ziegler, *General Pathology*, 1895.

upon the nature of the poison—*i. e.*, the variety of infecting organisms. The effects range from a slight increase of bodily temperature to a profound disturbance of the vital functions which ushers in death. They vary again from a transient intoxication to prolonged and constantly increasing evidences of poisoning.

Causes.—Many of the cases described under the head of fevers might be fitly included under the head of septic intoxication—indeed, fever is one of the prominent evidences of septic intoxication and their causes are similar. The causes of septic intoxication are the absorption from one or more foci of bacterial development of toxic substances which have been generated as the result of the vital processes of the organisms. Pathogenic organisms, in addition to local tissue-degenerations caused by their local action, induce these general symptoms of disorder by the character of the substances produced by them. Bacteria may gain entrance to the body at some point, as at a wound, be taken up from some portion of the alimentary tract or from the lungs, and while causing but comparatively slight local evidences of disease at the point of entry, may develop in other portions of the body and generate toxic substances in such situations. Miller[1] has recorded several forms of bacteria found in the human mouth which bring about pronounced septic poisoning when injected into the bodies of animals.

Symptoms.—The symptoms, both general and local, of septic infection, as stated, depend upon the character of the poison in the circulation—*i. e.*, by the variety of organism infecting. This may belong to any of the classes of bacteria capable of developing in any part of the body. It is only necessary that it be some form capable of transforming nitrogenous substances into simpler bases, which, if they gain entrance to the circulation from the tissues, alimentary canal, or respiratory tract, cause manifestations of the action of specific poisons. This is best illustrated by an examination of the products formed by the progressive decomposition of albumin. It is first peptonized, and, as pointed out by Brunton,[2] peptones directly injected into the blood cause poisoning. The substances formed in wounds, even those which go on to quick recovery, frequently cause an elevation of temperature when absorbed. The formation of compound ammonias (ptomaïns) is a succeeding stage of decomposition, in which such substances as sepsin, neuridin, tetanin, and ethyldiamin are formed, which are all active poisons. Next, simpler substances are formed, leucin and tyrosin, with the methyl-amins, and afterward such aromatic products as indol, the cresols, etc. Many of these substances may cause evidences of poisoning. Some of them are formed in the alimentary canal, some in the tissues, and if they are absorbed and not promptly eliminated,

[1] *Dental Cosmos*, Sept., Oct., and Nov., 1891. [2] Croonian Lectures, 1888.

cause toxic symptoms. Pathogenic bacteria, such as the typhoid bacillus, the bacillus of tetanus, the bacillus of diphtheria, and others, cause the formation of albuminous bodies, allied to ptomaïns, much more poisonous than the latter; these substances have been called toxins or tox-albumins (Brieger).

The general symptoms of septicæmia may be described as, first, affection of the nearest lymphatic glands, in which the organisms become lodged. Infective inflammation occurs; there is more or less fever, the height and character of which vary with the specific organism. In the more pronounced cases chills occur. There occur diarrhœa, and, as a rule, evidences of profound debility; the heart-action and the pulse become rapid and weak, a clammy skin is noted, and disorder of the central nervous system is present. If the infection occur from a wound or an evident focus of inflammation, the wound-discharges become putrid—*i. e.*, show evidences of progressive decomposition.

Treatment.—The first consideration in treatment is the removal of the cause if possible, and the disinfection of the local disease-focus. Failing in this, or coincidently with it, the principle of therapeutics is to sustain the vitality of the patient until the eliminative functions of the body have disposed of the poisonous substances and their generators (the micro-organisms). The general nutrition is to be supported by concentrated nutriments, beef peptonoids, protonuclein, sterilized milk, etc. The flagging heart-action is to be supported by stimulants—alcohol and strychnia. Brandy or whiskey is administered until the heart's action is found to grow more steady and vigorous. The intestinal tract is to be kept clear, and intestinal antiseptics administered—salol, naphthalin, and β-naphthol. The temperature is kept within bounds by large doses of quinine and cool sponging. The mouth is to be freely sprayed with strong antiseptics: hydrogen dioxid, listerine, formalin (1 per cent.).

Pyæmia.

The word pyæmia (Greek *puon*, pus, and *hæma*, blood) does not imply the presence of pus in the blood, as might be inferred, but is used to designate a condition in which there is widespread pus-formation in areas of the body, each suppurating focus being at some point of the circulatory system. Toxic substances are formed at such foci, and complicate a general septicæmia; the condition has been called septico-pyæmia.[1]

Causes.—Pyogenic organisms, exercising their specific action upon the walls of bloodvessels, cause inflammation of the walls and coagulation of the contained blood, which coagulum becomes infected by the

[1] Ziegler.

organisms. Portions of the coagulum become detached and are carried along the bloodvessels, setting up suppurative processes wherever the fragment happens to find lodgement. They are carried *via* veins to the lungs, and, stopping there, suppuration ensues; carried into the pulmonary veins, they reach the heart, where they may excite inflammation; carried into the arteries, each point of arrest becomes a centre of suppuration. These organisms may find their way into the circulation in the manner described from any area of suppuration in the body in which veins may be engaged.

Symptoms.—The symptoms of pyæmia are, in general, those of septicæmia, but, as a rule, their appearance is more delayed from the date of the reception of an injury or the outbreak of the primary suppuration. The onset of pyæmia is usually by a chill or a succession of chills. Each fresh area of pus-formation is believed to be announced by a chill and a rise of temperature. The temperature is subject to remissions, and sudden variations in its height are noted. The general symptoms are those of an adynamic fever. Local symptoms appear according to the point of lodgement of septic emboli. Pus-centres may be found in the lungs, and cause symptoms of dyspnœa; collections frequently occur in joints, causing loss of mobility; the swellings being apparent, eruptions appear on the skin; typhoid symptoms become more pronounced, and an increasing debility ushers in a usually fatal ending.

Treatment.—The treatment of pyæmia should be preventative. The carrying out of rigid antiseptic precautions has much lessened the frequency of pyæmia. If areas of infection are removable, they are removed no matter what extent of operation may be necessary. The general treatment is the same as in septicæmia, with much less hope of recovery.

A consideration of the infective surgical processes in connection with dental and oral diseases is of the utmost moment to the practitioner of dentistry. Nearly all of the diseases which the dentist is called upon to treat are infective from their inception. Moreover, the saliva, holding in suspension numerous forms of bacteria, both saprophytic and parasitic, and their waste, is a highly infective fluid.

It has been clearly demonstrated by the researches of Miller[1] that many forms of bacteria found in specific diseases, and found inhabiting the intestinal tract, are more or less constantly present in the human mouth, and that the pathway in many general infections is no doubt *via* the oral cavity. A wound made in the human mouth is necessarily an infected wound. In the vast majority of cases the body exercises its protective function in a phagocytosis,[2] which disposes of invading bac-

[1] *Micro-organisms of the Human Mouth.* [2] Hugenschmidt, *Dental Cosmos*, 1896.

teria. In other cases it is beyond question that this protective provision fails and infection occurs.

The principles, both pathological and therapeutic, which have been thus far expounded are the general principles upon which medical and surgical practice rests. They are in large part applicable to a proper and intelligent practice of dentistry. Dental practice is daily becoming more and more recognized as a special branch of surgery, the pathology, morbid anatomy, and therapeutics of which are modified by peculiarities of structure and position as in any other specialty. The origin, structure, and function of dental parts being special and distinctive, their diseases and the treatment of their diseases are also necessarily specialized. Restating that in order to comprehend the nature of morbid processes in a part it is necessary to have a comprehension of the embryology, histology, physiology, anatomy, and physiological chemistry of the part, the transition is now naturally to a discussion of these factors in so far as their connection with disease-processes may be clear.

SECTION II.

ANATOMY AND DEVELOPMENT.

CHAPTER VII.

THE DEVELOPMENT AND STRUCTURE OF THE JAWS AND TEETH.

Aberrations of Development.

For a proper comprehension of many of the abnormal conditions found associated with the jaws and teeth some familiarity with the embryology and an intimate knowledge of the histology of the parts are necessary preliminaries. The jaws and teeth, or the modification of such structures, play an important part in the economy of nearly every class of animals. The jaws, designed for the seizing of prey, represent the first structures concerned in the process of nutrition. Specialized structures with which the jaws are armed, teeth or their modifications, add to this primary armament and increase its usefulness. The teeth and jaws are so intimately associated that their embryology must be studied together.

Fig. 43.

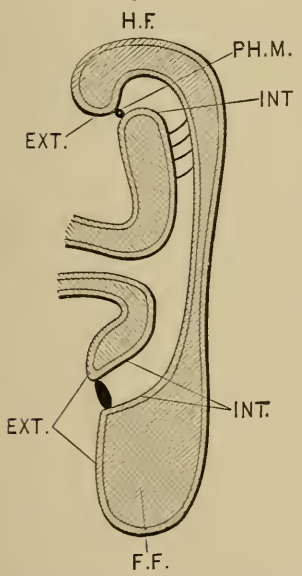
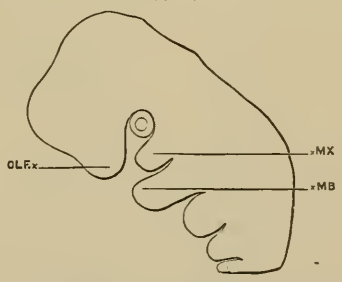


Diagram showing relations of epi- and hypoblast: H.F., head-fold; F.F., tail-fold; INT., hypoblastic layer of embryo; EXT., epiblastic layer; PH.M., pharyngeal membrane. (Hertwig.)

Fig. 44.



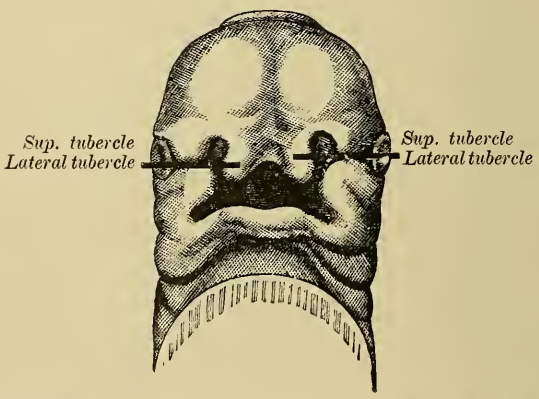
Diagrammatic outline of a human embryo of about seven weeks, showing the relations of the mandibular, MB, with the maxillary, MX, and olfactory processes, OLF. The maxillary process is seen as an outgrowth from the base of the mandibular process. (Allen Thomson.)

The embryology of the parts concerned with stomatology begins at

a very early period—before the twelfth day the future mouth may be located (His). The embryo, covered externally by an epiblastic layer, is traversed from near the tail-fold to near the head-fold by a tube lined with the hypoblast. At the tail- and head-ends of this tube the epiblast and hypoblast are fused together, forming septa; the upper septum separates the future mouth from the future pharynx (PH.M., Fig. 43). The face and jaws are outgrowths and attachments to the primitive cranium, represented by the head-fold. Before the fourth week of gestation there appear beneath the head-fold four pairs of buds growing toward the median line. From the uppermost of these growing processes (MB, Fig. 44) the lower jaw develops. From near the bases of these processes buds are given off, which grow obliquely forward and upward; these processes are the embryonic upper jaw (MX, Fig. 44). At the same time a knob-like process grows downward from the end of the head-fold, and by the time the lower maxillary processes have united in the median line a cavern is formed between these several processes, which will form the future mouth and future nasal cavities. This corresponds with about the fourth week of gestation (Fig. 45).

The globular processes growing downward form the intermaxillary processes from which the intermaxillary bones and the median portion

FIG. 45.



Head of an early human embryo, showing the disposition of the facial fissures and of the superior and lateral tubercles. (After His.)

of the lip develop. The upper maxillary processes grow forward and inward, fusing later with the intermaxillary processes of each side (Fig. 46, B). Each of these processes consists of a central mass of mesoblastic tissue—*i. e.*, tissue out of which the several connective tissues of the jaw will develop bone, cartilage, muscle, and the ordinary connective tissues.

As the superior maxillary processes grow forward to meet the descending intermaxillary processes, each sends inward a horizontal branch toward the median line, which progressively divides the large general

cavity into two compartments (N.C., nasal cavity, and O.C., oral cavity; Fig. 46, A), and when these horizontal processes fuse in the median line with the descending maxillary (intermaxillary) processes the nasal and oral cavities are separated. These fusions occur about between the seventh and ninth weeks of gestation, the union at the forward end occurring first and progressing backward, the septum being completed about the tenth or eleventh week. Malformations due to the non-union of the parts date, therefore, from this period. Several types of deformity arise from the non-union or imperfect union of these parts. The horizontal or palatal processes may unite with one another and with the intermaxillary processes, but their outer portions may fail to fuse upon one or both sides, constituting the condition known as harelip, shown in its extreme form in Fig. 47.

Fig. 46.

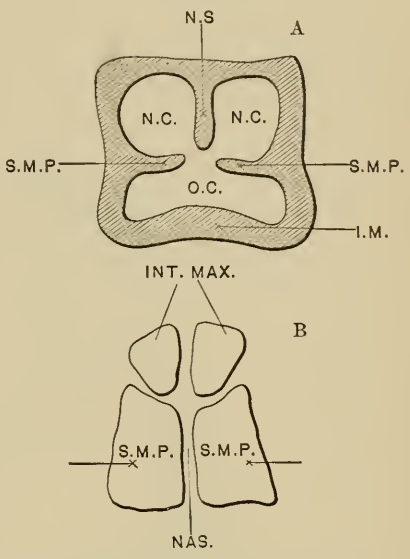


Fig. 47.

Complete bilateral fissures (coloboma) of face. (Guersant.)

If the horizontal plates fail to unite with one another, an opening between the nasal and oral cavities remains, the condition being called

cleft palate. The intermaxillary processes may fail to unite upon one or both sides with the superior maxillary processes proper, forming clefts which extend obliquely from the median line. As a rule, the outer or lip portions of these processes also fail to unite in this latter condition, so that oblique palatal clefts and harelip are commonly associated. If the right and left globular or intermaxillary processes

FIG. 48.

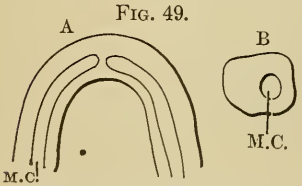
Median fissure of the lower lip and chin. (Marshall, after Wöfler.)

fail to unite with one another, a fissure will exist in the median line of the lip. It is rare that the right and left inferior maxillary processes fail to unite; the condition is, however, occasionally seen (Fig. 48).

DEVELOPMENT OF THE LOWER JAW.

The inferior maxillary processes have united in the median line beneath the partially developed buds of the superior and intermaxillary processes. The central portions of the mesoblastic tissue composing the bodies of these processes become transformed into two rods of cartilage, which act as supports to the arch during the period in which the inferior maxillary bone is forming. The cartilages of the right and left sides do not fuse together at the future symphysis (Hertwig) (Fig. 49). This cartilage is but a temporary structure; it undergoes atrophy at about the sixth month of gestation, and at birth but few fragments are found near the symphysis. The end of the

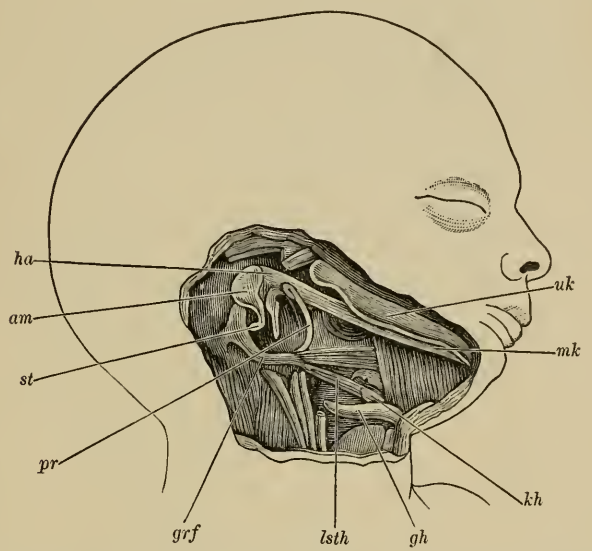
FIG. 49.



Showing Meckel's cartilage (M.C.) in longitudinal and transverse section.

cartilage in the base of the inferior maxillary process becomes the future malleus (one of the bones of the middle ear) (Fig. 50). The portion of

FIG. 50.



Head and neck of a human embryo eighteen weeks old, with the visceral skeleton exposed. The lower jaw is somewhat depressed in order to show Meckel's cartilage, which extends to the malleus. The tympanic membrane is removed and the annulus tympanicus is visible. *ha*, malleus, which passes uninterruptedly into Meckel's cartilage, *mk*; *uk*, bony lower jaw (dentale), with its condyloid process articulating with the temporal bone; *am*, incus; *st*, stapes; *pr*, annulus tympanicus; *grf*, processus styloideus; *lsth*, ligamentum stylohyoideum; *kh*, lesser cornu of the hyoid bone; *gh*, its greater cornu. Magnified. (After Kölliker.)

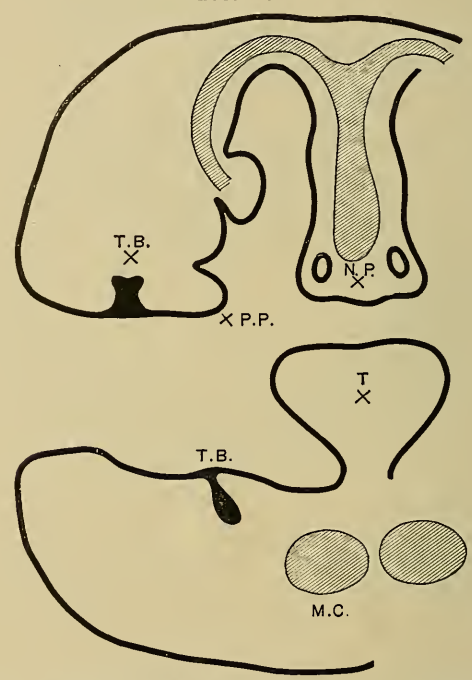
the cartilage running from the malleus to the formed bony lower jaw becomes transformed into the internal lateral ligament of the inferior maxilla (Hertwig).

DEVELOPMENT OF THE TEETH.

The first evidences of tooth-formation are seen at about the sixth week of gestation, at a period when the superior and inferior maxillary processes are but ill-defined masses of mesoblastic tissue surrounded on all sides by epiblastic tissue. Before the union of the processes which are to separate the nasal from the oral cavity and which form the future palate, is complete, the first evidences of tooth-formation may be observed. It is to be borne in mind that during the entire period of tooth-formation other formative changes are in operation, out of which arise all of the parts associated with the teeth. A transverse section of the lower jaw at this period will exhibit an ellipsoidal surface in which there can be plainly seen a mass of indifferent mesoblastic tissue, surrounded by epiblastic tissue, except at the middle, where it is reflected over a pear-shaped structure—the future tongue (Fig. 51). Within the substance of

the mesoblastic tissue are seen two elliptical areas, the sections of Meckel's cartilage. If the sections are made near the median line, these oval areas will be close together; if much farther back, they will be widely separated. On the upper surface of the jaw, upward and outward from Meckel's cartilage, the epithelium is seen to be much thicker at a point on each side than it is over other parts; the free surface of the epithelium at this point rises above the general surface, and where the epithelium is in contact with the mesoblastic tissue it sinks below the general

FIG. 51.



A section through the developing jaws at about the seventh or eighth week: N. P., nasal process; T., tongue; M. C., Meckel's cartilage; T. B., tooth-bands.

surface (Fig. 51, T.B.). Sections made from before backward show that this dipping in and elevation of the epithelium extend along the arch of the jaw, so that if viewed from above it presents a horseshoe-shaped ridge occupying the summit of the embryonic jaw. The elevation is greatest in front, thinning gradually toward the heels of the horseshoe. This is the primary dental ridge. Older microscopists, notably Goodsir, obtained specimens from which the epithelium had been removed, and hence the depression made by the base of the epithelial growth in the mesoblastic tissue gave the appearance of a groove, called by Goodsir the primitive dental groove. Sections made of older jaws exhibit the gradual evolution of the structures of the jaw.

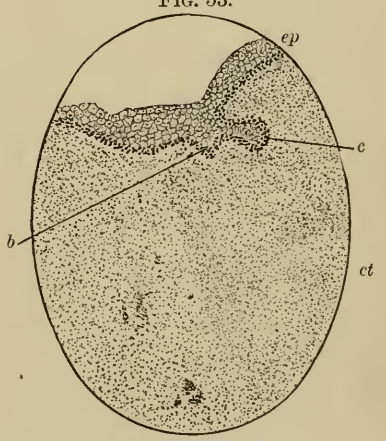
The local overgrowth of epithelium proceeds until the ridge is well

FIG. 52.

Longitudinal transverse section of the inferior maxilla of a porcine embryo: *b*, band, solid at anterior portion, but divided posteriorly into band and lamina. (3 cm. × 40.) (Sudduth.)

marked, and the lower epithelium—*i. e.*, the germinal layer—is seen to sink deeper and deeper into the mesoblastic tissue, forming a continuous horseshoe-shaped band of epithelium; the epithelium in this condition is called a band (Fig. 52, *b*). At a later stage the rounded base of the band becomes flattened; this base is then termed a lamina—the dental lamina. From the inner angle of this lamina ten buds are given off, each corresponding with a future deciduous tooth (Fig. 53, *c*). These ten buds, or, as they are called, dental cords, grow inward into the mesoblastic tissues, the anterior preceding in growth the posterior cords. Each developing cord appears soon to meet with an outlined resistance, corresponding roughly with the shapes

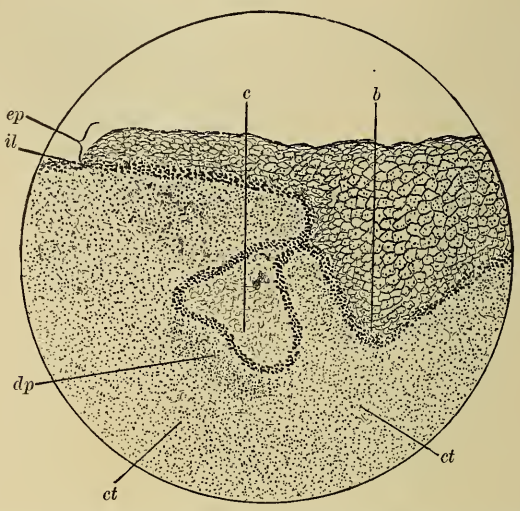
FIG. 53.



Vertical section through band from jaw of porcine embryo: *ep*, epithelium; *b*, band: *c*, cord: *ct*, connective tissue. (3½ cm. × 60.) (Sudduth.)

of the several teeth. It might be said with perhaps equal propriety that the base of the cord assumes these shapes, as the origin of the assumption of the typical forms is unknown. The mesoblastic tissue

Fig. 54.



Vertical section through band and cord of 3½ cm. porcine embryo × 60: *ep*, epithelium with infant layer (*il*); *b*, band; *c*, pear-shaped cord; *dp*, dental papilla; *ct*, connective tissue. In this cut the walls of the cord are shown very plainly to be a continuation of the infant layer of the epithelium. (Sudduth.)

is observed to be condensed, to be faintly differentiated, from the indifferent mesoblastic tissue; this condensed area will ultimately form the dental pulp (Fig. 54).

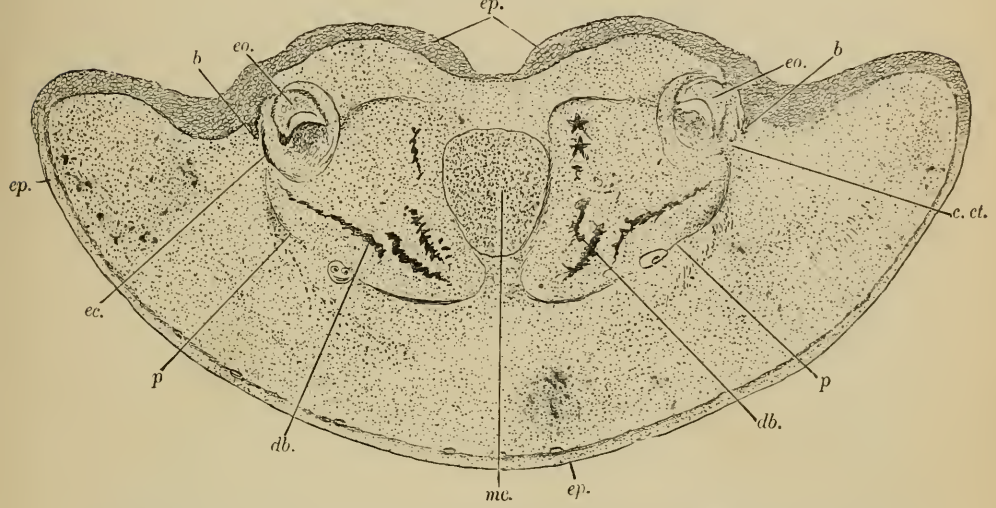
As the ingrowth of the epithelium proceeds, definite changes are seen to occur in the epithelial mass, which will later be described in detail. This epithelial mass is the structure through which the enamel of the teeth will be formed, and hence is called the enamel-organ. As it grows bodily inward, changes are observed in the tissues about it; the indifferent mass of mesoblastic tissue, constantly increasing in size, is seen to undergo differentiations at isolated points; bone-forming cells—osteoblasts—make their appearance and form bone without antecedent cartilage and without evidence of a periosteum; this has been called interstitial bone-formation[1] (Fig. 55). Condensations of tissue occur about the outside of these several bone-islands, an embryo periosteum; the development of the bony jaw is now in progress. It will be observed that Meckel's cartilage is not included in the area marked off as the bony jaw (Fig. 55).

Outside the embryo periosteum evidences of the future muscles of the jaws appear. As the growth of the enamel-organ continues, and as

[1] Sudduth, in *American System of Dentistry*.

it assumes upon its inner surface the form of the future tooth, the connective tissue surrounding it acquires the character of a thick fibromembrane, which later encloses the entire developing tooth-pulp and enamel-organ. These structures, out of which all of the dental tissues, with the exception of the nerves, will be evolved, are now collectively called the dental follicle. The growth of bone continues, so that the

Fig. 55.



Vertical transverse section of jaw of porcine embryo, showing differentiation of periosteum: *pp*, periosteum of either jaw; *c.ct*, follicular wall, appearing as a continuation of the periosteum; *b*, band; *eo*, enamel organs for premolars; *ep*, epithelium; *db*, developing bone; *mc*, Meckel's cartilage. (5½ cm. × 25.) (Sudduth.)

ten follicles lie now in a gutter of bone, the developing maxilla. Before the enamel-organs assume their typical forms the cords from which each arose are each seen to give off a bud, which grows downward and inward; as regards the first cords, they are cords out of which the enamel-organs of the permanent teeth will be developed. The cord of each temporary tooth gives off the cord of its permanent successor. Behind the cord for the second deciduous molars, and some writers have said directly from it, a cord arises for the first molar of the permanent denture; this occurs before the sixteenth week. At about this period the epithelial bands connecting the enamel-organs with the surface epithelium of the mouth break up into whorls and connection between the developing teeth and mucous membrane is lost.

Development of the Enamel.

Each of the dental tissues is developed after a distinctive manner, and the process requires close observation with very high microscopic powers to make out all of the details. Although in point of time the

first layer of dentin is deposited before the first layer of enamel, the changes and evolutions which occur in the enamel-organ may be made out before the details of the structure of the dental pulp become apparent; for this reason the development of enamel is first studied.

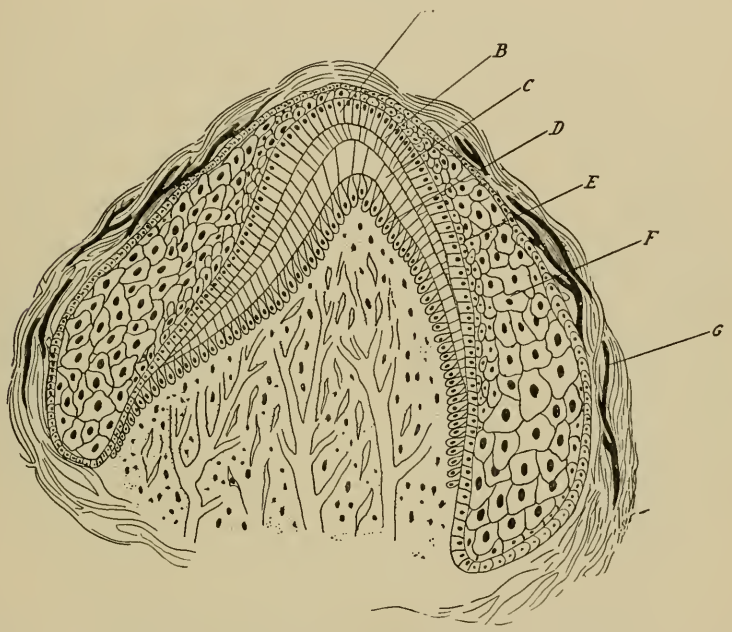
Fig. 56.

Vertical transverse section of jaw of porcine embryo, injected: *ep*, epithelium, with (*il*) infant layer; *a*, layer of ameloblasts; *o*, layer of odontoblasts; *cp*, cord for permanent tooth; *ot*, outer tunic; *it*, inner tunic; *sr*, stellate reticulum; *wh. ep.*, whorls of epithelium formed from outer tunic and stellate reticulum; *d*, dentin; *dp*, dentinal pulp; *v*, bloodvessels of pulp; *ct*, connective tissue; *c. ct.*, follicular wall; *p*, periosteum; *sp*, space. (10 cm. × 60.) (Sudduth.)

It will be observed that the indipping of epithelium into the mesoblast is an indipping of the deepest or germinal layer of epithelial cells. It is noted at this period that while the epiblastic tissue is clearly

marked off from the mesoblastic, no such structure as a basement-membrane exists. As the dental cord increases in depth it increases in breadth, the germinal layers remaining distinct; older cells make their appearance between the layers. Following the formation of the cords and their evolution into the enamel-organs, it is seen that the organ becomes first conical, the sides and base of the cone being formed of germinal epithelium; a depression constantly increasing in depth forms in the base of the cone, until finally the enamel-organ is seen to consist of a double layer of germinal epithelium, the layers being separated by epithelial cells of an older type, which undergo

FIG. 57.

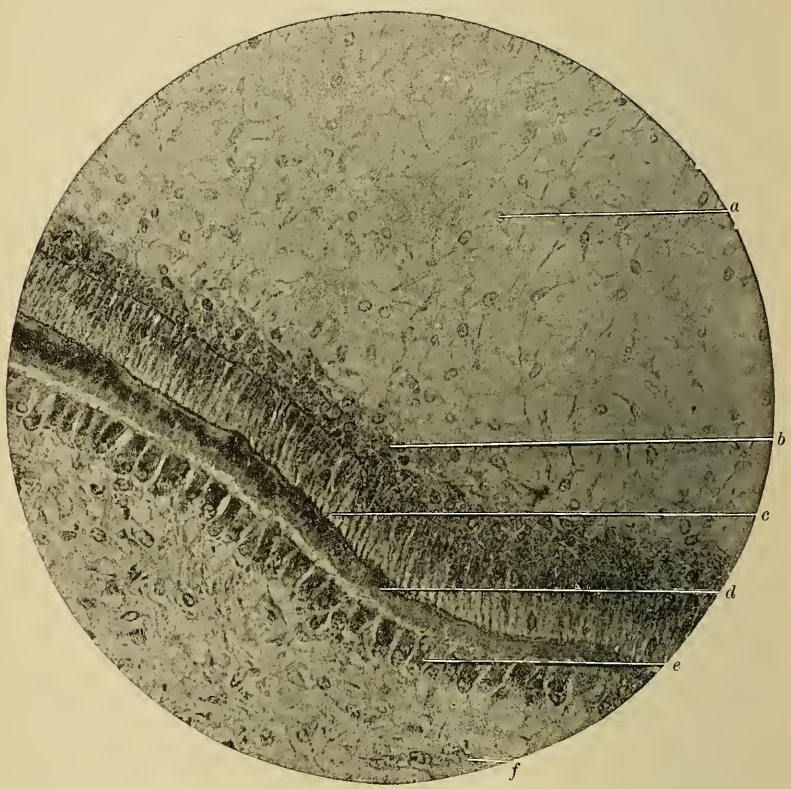


Section of a developing cuspid (human) at about the sixth month: *A*, ameloblasts; *B*, enamel; *C*, dentin; *D*, odontoblasts; *E*, stratum intermedium; *F*, stellate reticulum; *G*, follicular wall.

a remarkable increase in size. At this time, or a little later, blood-vessels make their appearance in the condensed mesoblastic tissues covered by the enamel-organ. Springing from the base of this condensed mass, fibrous tissue makes its appearance, extending up and along the sides of the enamel-organ; this is recognized as the wall of the dental follicle (Fig. 56, *c.ct.*). Between the fourth and sixth months of gestation the differentiation of the parts of the enamel-organ are evident. The cells of the inner germinal layer of epithelium, that next to the mesoblastic tissue, are seen to assume a prismatic form, the change being most pronounced above the apex of the future pulp. The outer

epithelial layer for the present is of cuboidal cells ; between these two layers two types of epithelial cells are seen. The cells occupying the greater mass of the enamel-organ are greatly distended ; owing to the appearance of these cells when seen in section (Fig. 57, *F*) the structure has been called the stellate reticulum, or star-like network. It was at one time thought that the cells of this portion of the enamel-organ were devoid of cell-contents, the distention being caused by the accumulation of fluid between the cells. It has been lately shown [1] that the cells themselves are enormously distended or enlarged (Figs. 57, 58). The layer

Fig. 58.



Section of developing tooth of an embryo calf: *a*, stellate reticulum of enamel-organ; *b*, stratum intermedium; *c*, ameloblasts; *d*, dentin; *e*, odontoblasts; *f*, bloodvessel—corpuscles *in situ*. × 275. (Williams.)

of cells next to the pulp which have assumed the prismatic form are those through which enamel-building will be accomplished, hence they are called ameloblasts or enamel-builders. Between the ameloblasts and the stellate reticulum proper, the epithelial cells are of smaller size than those of the latter, are distinct, and are more firm. This layer is called,

[1] J. L. Williams, *Dental Cosmos*, 1896.

from its position, the stratum intermedium. The fibrous tissue forming the follicular wall is observed in close relation with the outer epithelial layer of the enamel-organ. The structures necessary to the formation of enamel are now differentiated, but before this process can be comprehended a brief survey of the physiological chemistry of the operation is advisable.

The enamel of the teeth, by present methods of chemical analysis, is found to be composed in large part of calcium salts, the phosphate and carbonate; and yet in its structure and texture it in no way resembles the ordinary crystallized forms of these mineral salts, so that the formation of enamel is not merely the deposition and crystallization of calcium phosphate and carbonate. It is beyond question that enamel is a compound of organic origin, and the nature of its substance must be sought for among the organic compounds.

The experiments of Harting, Rainey, and Ord,[1] have shown a reaction which no doubt has a direct bearing upon the formation of all calcified tissues. If to an albuminous solution a solution of a calcium salt be added, the calcium enters into chemical combination with the albumin, forming a substance indefinitely known as albuminate of calcium, and called by its discoverer calco-globulin. If calcium carbonate be formed in a solution of albumin, the above combination occurs, making definite structural forms, minute laminated spheres, which are called calco-spherites; these spheres coalesce and form laminated masses—*i. e.*, form in layers. When exposed to the action of dilute acids these spherites are more resistant than the crystallized salts; moreover, after the action of the acid the form of the spherite remains. This chemical fact, the union of crystalloidal with colloidal substances, is, no doubt, of wide significance in general and special pathology, for it is extremely probable that the formation of all pathological concretions is an expression of some such reaction. The evidence is strong that calcium albuminate is the basis of all of the calcic tissues; but precisely where its formation occurs in enamel-formation is unknown; presumably, it occurs or is completed in the enamel-forming cells.

Williams has shown,[2] by selective staining, that prior to and during enamel-formation the ameloblasts are separated from the developing enamel upon one side and from the stratum intermedium upon the other side by what appear to be membranes, so that any future generation of enamel-cells, if such occur, must be from the ameloblasts themselves. Moreover, the cells of the stratum intermedium are not of the germinal type. Andrews[3] calls attention to the fact that if a sec-

[1] R. R. Andrews, *American Text-book of Operative Dentistry.*

[2] *Dental Cosmos*, 1896, p. 107 *et seq.*

[3] *American Text-book of Operative Dentistry*, 1897.

tion of an enamel-organ at this period be placed under a cover-glass and a diluted mineral acid be permitted to run under the glass, bubbles of gas (carbon dioxid), are disengaged, showing the presence of calcium salts in the structures. The bubbles form in the stellate reticulum, in the ameloblasts, in the structure between the ameloblasts and the first-formed layer of dentin, and in the latter tissue.

In the ameloblasts, having large, well-marked nuclei at their distal

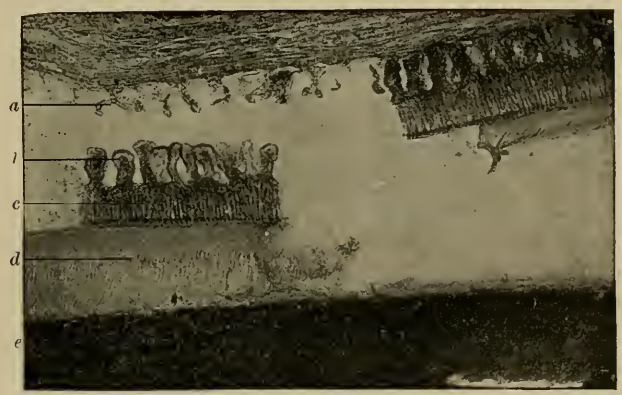
FIG. 59.

Section of developing tooth of an embryo calf: *a*, *b*, nuclei of reticulum of enamel-organ, showing spongiose character; *c*, outer ameloblastic membrane; *d*, inner ameloblastic membrane; *e*, *f*, enamel-globules faintly showing nuclear network. × 1000. (Williams.)

portions two distinct cell-contents are seen: one, glistening droplets of various sizes, which coalesce, becoming larger as they approach the proximal end of the ameloblasts, out of which they are extruded against the forming dentin. In addition to these droplets the ameloblasts are

seen to contain one or more globular bodies, all of like size, lying between the nucleus and the proximal end of the cell. These globules are connected with one another by plasmic strings. Into the mass formed by the fusing together of the droplets first extruded from the ameloblasts, these uniform-sized globules are deposited. The first-named material, nominally calco-globulin, is a cement-substance which flows around and about the globules and their processes. The globules, from mutual pressure, assume naturally a prismatic form. The globular bodies are called by Williams enamel-globules. The small droplets out of which the cement-substance is formed are found also in the stratum intermedium; differential staining demonstrates this. The enamel-globules stain differently, are of one size, and are only noted between the nucleus and the proximal end of the cell. Williams infers that they arise by a process of mitosis or cell-multiplication from the nucleus itself, a most rational conclusion. As soon as the first layers of enamel have formed a notable change is seen to occur in the enamel-organ; the stellate reticulum disappears over the forming enamel. The calcic material stored in its cells has been exhausted in forming the first layer of enamel; the succeeding enamel has a different

FIG. 60.

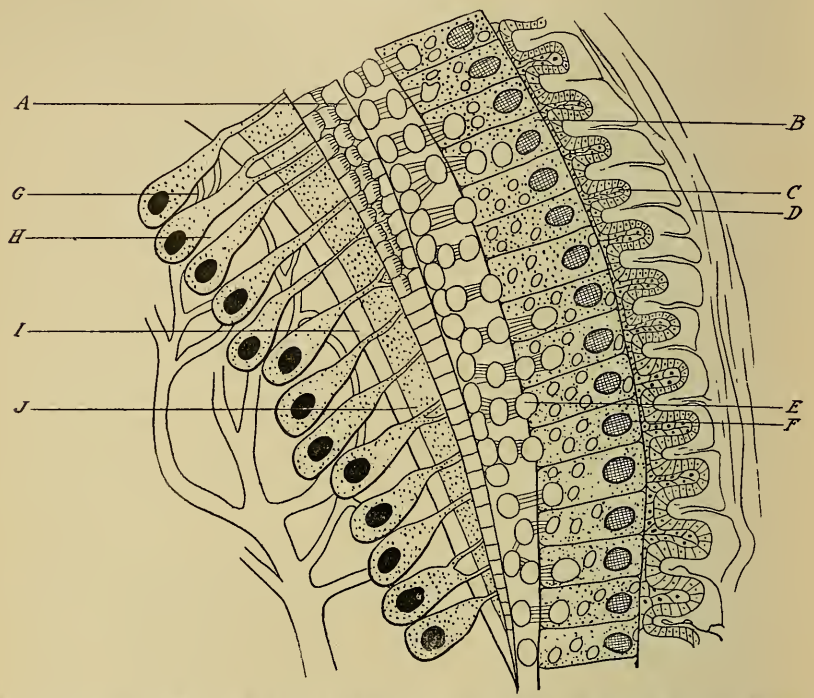


Section of incisor of rat: *a*, capillary loops torn out of secreting papillæ; *b*, secreting papillæ after removal of capillary loops; *c*, ameloblasts; *d*, enamel; *e*, dentin. × 80. (Williams.)

source of formative material. The stellate reticulum atrophies and the outer boundary wall of the enamel-organ comes in apposition with the stratum intermedium. Williams observed in the enamel-organs of rodents (Fig. 60) that the cells of the stratum intermedium become arranged over loops of vessels from the vascular coat, so that papillæ are formed. He infers that a similar arrangement occurs in the enamel-organ of man; and that the function of the papillary structure is the selection from the blood-plasma of material to be passed into the ameloblasts out of which

the enamel is formed. The deposition of cementing-substance and connected globules continues, and the enamel increases in thickness from the dentin outward (Fig. 61). The enamel over the tips of the

FIG. 61.



Mode of enamel-deposition: *A*, formed enamel; *B*, ameloblasts; *C*, secreting papillæ of stratum intermedium: *D*, bloodvessels in external fibrous coat and to secreting papillæ; *E*, enamel-globules with connecting plasmic strings; *F*, nuclei of ameloblasts; *G*, odontoblasts; *H*, blood-supply to odontoblastic layer; *I*, unformed dentin: *J*, formed dentin. Semi-diagrammatic. (After Williams.)

teeth is first formed, and as this portion of enamel increases in thickness successive portions of the ameloblastic layer acquire formative function; the covering stellate reticulum gradually disappears, until by the time the neck enamel begins to form no vestiges of the stellate reticulum remain. This deposition of substance continues until the crown of the tooth has its normal form, the ameloblasts appearing to undergo partial calcification themselves, resulting in the formation of a continuous sheet covering the enamel, and constituting, at least in part, Nasmyth's membrane.

FORMATION OF DENTIN.

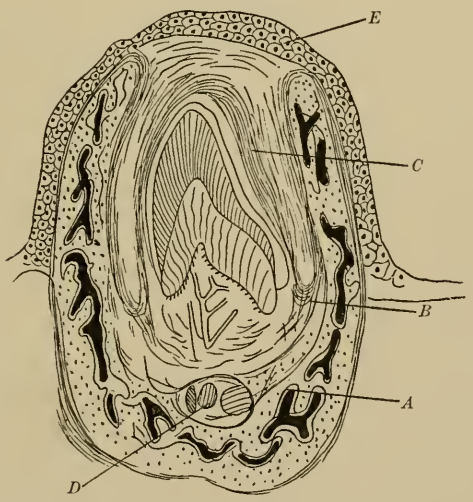
Prior to the appearance of any calcic tissues in the teeth, it is noted that upon the periphery of the developing pulp, notably in the positions of the future cusps, a layer of cells becomes differentiated from the in-

different mesoblastic tissues of the pulp; these cells assume an arrangement like that of cylindrical epithelium, but are not in lateral contact with one another. Beneath this layer of cells, called from their function the odontoblasts (*odous* and *blastos*), a capillary network is formed, the capillaries extending by loops into the odontoblastic layer (Fig. 61). Before any deposition of enamel occurs it is seen that the odontoblasts exude and extrude from their distal ends masses of calcospherites. As this deposition proceeds the odontoblasts recede, each cell leaving one or more branched processes in the dentin-deposit. The deposition continues, the dentinal pulp constantly decreasing in volume as the dentinal deposit increases in amount. When it is remembered that the deposit of dentin begins before that of the enamel, it explains how the processes from the odontoblasts may be found in the enamel in exceptional cases; the processes may grow outward between the ameloblasts, and a deposit of enamel occur about them. This condition, normal in the teeth of some animals, is occasionally seen in the teeth of man.[1]

Formation of Cementum and Roots of Teeth.

The deposition of cementum begins after the formation of enamel is practically complete. The formation of cementum is identical with the

Fig. 62.



A, developing bone; *B*, tissue reflected from follicular wall and forming alveolar periosteum; *C*, follicular wall; *D*, vessels and nerves; *E*, epithelium of gum.

subperiosteal formation of bone. By the time that enamel-formation is complete, or nearly so, the greater bulk of the crown dentin has been

[1] Williams, *Proc. Odontological Society of New York*, 1896.

formed and the dental pulp is contracted from above, less upon the sides, and is widely open beneath. The developing teeth at this stage rest in a gutter of bone, which at present makes up the greater bulk of the inferior maxilla. The bases of the teeth are not far removed from the canal containing the nutrient vessels and nerves; the inferior dental canal—that is, the neck of the tooth—is in the position which will be occupied by the apex of the root after the tooth is fully formed. It is evident, therefore, that the growth of the root of the tooth must mean its extension upward, the only direction in which it is free to move, it being observed that the gutter of bone in which the teeth lie is not completely closed above (Fig. 62). It is asserted by Hertwig[1] that the extremity of the enamel-organ grows downward beyond the crown of the tooth, so that the following relationship is established: a layer of odontoblasts is found upon one side of this epithelial layer; upon its other side the follicular wall comes in close apposition. The epithelial sheath atrophies, and is probably the source of the epithelial whorls found in the pericementum. The deposition of root-dentin begins before any cementum is formed, the first deposited portions of dentinal matter being frequently marked by faulty organization, forming the line of imperfect calcification known as the stratum granulosum. The fibrovascular covering of this portion of dentin becomes a modified periosteum; a layer of osteogenetic cells appears upon the portion next to the dentin. These cells, from their function, are called cementoblasts; they lie as flattened cells, between which large fibres from the fibrovascular portions of this tissue pass. The fibrovascular osteogenetic tissue spreads over the developing bone about the forming tooth, and is thus both alveolar periosteum and pericementum. The crown of the tooth is thrust forward as the root develops, and no doubt the gradual growth of the bone about the teeth is also a factor in the movement of the tooth. The remainder of the process will be discussed under the head of dentition.

By the time the crowns of the teeth have made their appearance through the gum the root-formation is still incomplete. As the crown of the tooth advances the bone which overlays the crown undergoes sufficient absorption to permit the passage of the crown, the bone being at other parts separated from the tooth and its partially formed root by the thick layer of connective tissue, forming pericementum and alveolar periosteum. Deposition of dentin and cementum continues until the root-form is complete; after eruption, bone-formation about the tooth reduces the thickness of the pericementum and sheaths the roots of the teeth in a bony covering.

The development of the permanent teeth follows a similar course;

[1] Röse, *Dental Cosmos*, 1893.

beginning with the follicle of the first permanent molar, which is differentiated between the fourth and fifth months of gestation, situated behind the developing deciduous second molar. The enamel-organ of the second permanent molar arises from the cord of the first, between the seventh and eighth months of gestation; the enamel-organ of the third permanent molar, arising in its turn from the cord of the second molar, does not appear until about the third year. The cords for the other permanent teeth—incisors, cuspids, and bicuspids—appear as offshoots from the cords of the temporary teeth which they are to succeed, at from the fourth to the fifth month. At the time of birth the follicles of these teeth are complete, and calcification begins shortly thereafter. The development of the temporary teeth is accomplished in a period averaging about three years; that of the permanent teeth requires for the first formed tooth about seven years, and for the last formed about fourteen years.

Fig. 63.

Showing the relation of permanent tooth-follicle to the root of the temporary tooth.

The developing permanent teeth are situated behind and beneath the temporary teeth (Fig. 63), the development of bone about the follicles of the permanent teeth enclosing each of them in a distinct pocket of its own, a bony lamina existing between each follicle and the tooth-root adjacent. It is to be noted that the developing permanent teeth are separated from the inferior dental canal by a lamina of bone; the canal, it will be seen, has in the child at birth but comparatively little thickness of covering-bone beneath it—*i. e.*, the body of the bone is very slight.

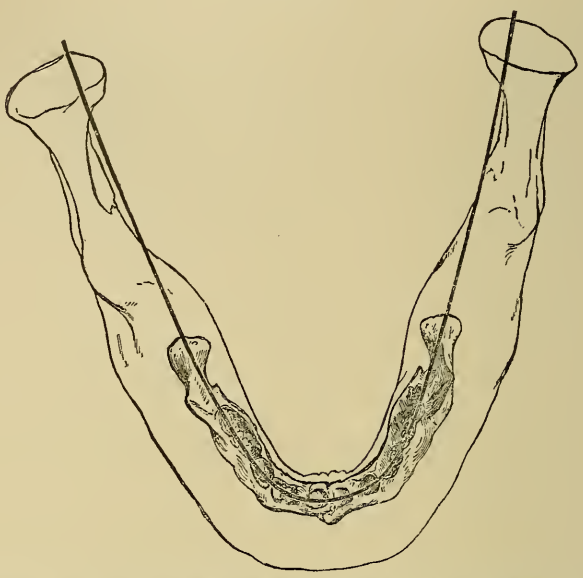
Later Development of the Maxillæ.

The maxillæ have developed during the period included in the foregoing description mainly as tooth-carriers and supports; the maxillæ proper have not developed to a corresponding degree. The body and rami of the lower jaw represent only the lesser portion of the entire maxillæ; the body of the upper jaw is at all aspects but a diminutive of the mature jaw.

At the time of birth Meckel's cartilage has disappeared, but a few fragments are left about the symphysis. The site of this anatomical scaffolding is represented by the mylohyoid groove, a groove beneath the inferior dental canal, which lodges the mylohyoid branch of the inferior maxillary nerve. As stated, the alveolar portion of the jaw forms its major and most prominent part. The body and rami of the bone are at a very immature stage. The body of the bone, that beneath the alveolar portion, is a comparatively small and thin shell of bone, something more than sufficient to accommodate freely the inferior dental artery,

nerves, and veins which it encloses. The rami of the jaw are very short. The coronoid process arises to but a small height, and almost

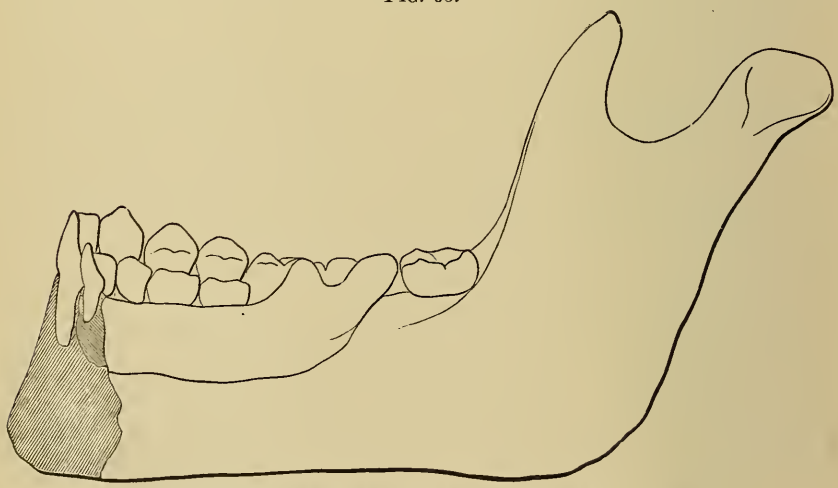
FIG. 64.



Representing a jaw of a nine months' fœtus, superimposed on an adult's jaw, to show in what directions increase has taken place. (Tomes.)

immediately behind the covering of the second deciduous molar follicle; the condyloid process is at about the same height, but little above the

FIG. 65.



Showing the relative sizes of jaws at the age of two years and in the adult.

level of the gum-summit, there is not that sharp distinction between ramus and body seen at later stages.

If the inferior maxillæ of an infant at the time of first dentition be compared with that of an adult, it will be seen that the radii of the dental arches in both jaws are nearly alike. The comparison may be more readily made if the two jaws be compared at the completion of the primary dentition (Figs. 64 and 65). It will be observed that the body of the mature jaw projects very much at the anterior and lateral aspects of the jaw, but the greatest amount of growth is backward and upward; these are, therefore, the directions in which the greatest growth occurs. The coronoid process, which at two years is but little behind the second temporary molar, recedes, until at about the age of seventeen years the space between the ramus and the former position of the second deciduous molar is sufficient to accommodate the three largest teeth of the dental series.

The depth of the jaw, the portion of the jaw lying beneath the mental foramen, is nearly acquired by the seventh year; the height of the jaw (the portion above) is not acquired until some time after the full eruption of the permanent teeth. From the time of birth, the anterior portion of the body of the jaw—that in front of the mental foramen—develops, as shown in Fig. 64, by additions to its front wall. The notable increase is backward and by an upward movement of the rami. The direction of the movement of the condyle during development is obliquely backward and upward. "The condyle occupies during development of the jaw, successively, every point of the internal oblique line."[1]

"The course of the coronoid process is to be seen in the external oblique line."[2] The formation of the condyloid portion of the bone is largely cartilaginous, the formation of the other growing portions of the bone being almost entirely subperiosteal. There is a complex accompaniment of resorption with deposition, in the formation of the rami of the lower jaw. The amount of bone deposited during the growth of the ramus of the jaw would represent a quadrangular block the thickness of the condyle, and extending backward from the posterior surface of the second bicuspid to the back edge of the jaw, and from the alveolar border to the height of the condyle at maturity; but as the deposition of bone occurs backward and upward, the redundant deposit undergoes an absorption which carves the bone into its typical form.

Development of the Upper Jaw.

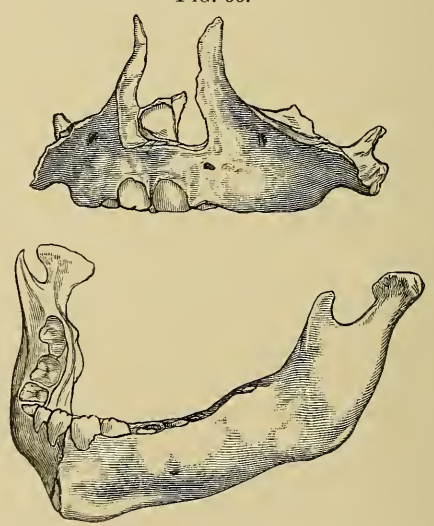
Observations as to the exact mode of development of the upper jaw are not so complete as those relating to the lower jaw. At the beginning of tooth-formation in the upper jaw the junctions between the right and left palatal processes, and of the intermaxillary processes

[1] C. Tomes. [2] *Ibid.*

with both, have not occurred. In the intermaxillary portions the follicles for the incisor teeth are formed. After the junction of the several processes the palatal curtain becomes differentiated into two parts: the anterior undergoes ossification; the posterior remains as a movable curtain—the soft palate. There occurs an interstitial formation of bone outlining the dental portion of the superior maxillæ, and soon a periosteum appears as in the lower jaw.

At the time of birth the upper jaw is at a stage of immaturity corresponding with that of the lower jaw. The partially formed crowns of the temporary teeth, the incisors, farthest advanced in development, next the first molars, then the cuspids, and finally the second molars, are all enclosed in a hollow arch of bone, having transverse divisions between each tooth; the spaces, or, as they are called by Tomes, loculi of the several teeth, are outlined in the degree of the tooth development (Fig. 66). Behind the second molar is the developing

FIG. 66.



Jaws of a seven months' child. The incisors in both maxillæ are being erupted by the absorption of the gum from their cutting edges and the elongation of the roots by calcification. (Tomes.)

first molar of the permanent denture; as in the lower jaw, the dental portion of the bone predominates. Taking the infra-orbital foramen as a fixed point of measurement, the top of the alveolar arch is but a short distance from the floor of the orbit. At the time of birth the roof of the mouth is but slightly arched from side to side. We may regard, as in the lower jaw, the main portion of the superior maxillæ at this stage of development to be a dentale, an alveolar portion; and that the body of the bone, as in the lower jaw, is in a fœtal condition; but when it is observed that the developing teeth are almost

on a level with the palatal processes, it is evident that these teeth are lodged in the maxillæ proper, and that the course of development creates the major portion of the alveolar bone subsequently. This is notably true of the follicles of the permanent cuspids, which occupy a position high up and behind the roots of the temporary tooth. A pair of dividers having one point in the infra-orbital foramen, and the other measuring from that point to, first, the symphysis edge, and next to the posterior alveolar edge will show, if applied to jaws of successive ages, a lengthening in both directions. The increase is most notably downward. The teeth shift their relations not only to their early environment, about the level of the palatal vault, advancing toward the alveolar border; but, in addition, the developing alveolar process lengthens the distance from the alveolar border to the infra-orbital foramen, not alone the alveolar process as generally understood, but the alveolar segment of bone itself increases in size, until the bone has the dimensions found in the adult. Measurements at this time show that the distance from the infra-orbital foramen to the alveolar border is greater than the depth of alveolar process developed.

Histology of the Mature Teeth.

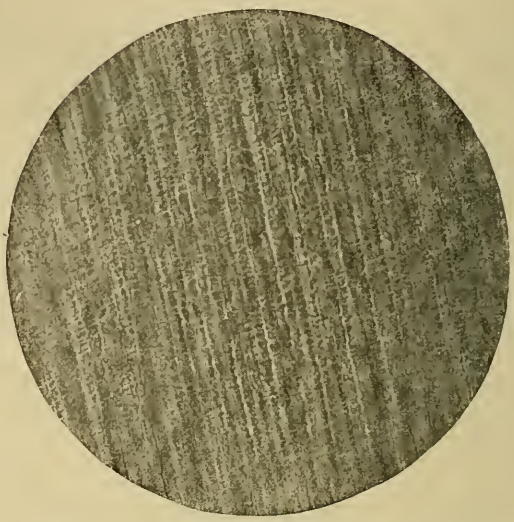
It is only through a study of the embryology of the teeth that the histology of their tissues becomes clear, and following the course of their development fully explains the structure of enamel, dentin, and cementum.

ENAMEL.

Sections of enamel show the tissue to be apparently made up of hexagonal prisms, which until recently were believed to be homogeneous throughout their length. Upon the theory that enamel consisted of the calcified bodies of the ameloblasts, hexagonal prismatic epithelial cells, there could be no other deduction; but the researches of Andrews, who demonstrated the interlacing basement-stroma of enamel, and the later work of Williams, which showed the duality of enamel-substance, render untenable the theory of continuous, homogeneous prisms. The final blow at the direct calcification theory, it will be recalled, is that the axes of enamel-rods and of the ameloblasts are at wide variance. An optical analysis of mature enamel in properly prepared specimens shows clearly the presence of two substances (Fig. 67). The clear spaces in the section are the cementing-substance binding together the calcified globules, shown dark in the section. It will be seen that these globules are superimposed upon one another in such a manner as to form rods marked by transverse lines. These rods were formerly called enamel-prisms. If a section be subjected to the action of a dilute min-

eral acid (HCl), the cement-substance between the rods and between the individual globules dissolves more rapidly than the substance of the globules, and there is produced the beaded appearance shown in A,

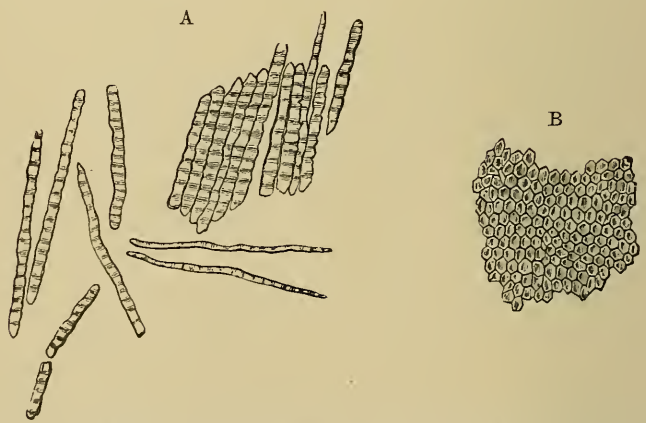
FIG. 67.



Section of enamel of human tooth. Photographed with Zeiss apochromatic lens and Powel and Leland apochromatic condenser. The optical parts accurately centred and the focus "critical." The enamel-rods are seen to be resolved into distinct sections (enamel-globules), the cement-substance often passing entirely between the sections. ×400. (Williams.)

Fig. 68. Sections cut transversely to the long axis of the rods exhibit the appearance shown in B, Fig. 68. The action of the dilute acid

FIG. 68.



Enamel-prisms: A, fragments and single fibres of the enamel isolated by the action of hydrochloric acid; B, surface of a small fragment of enamel, showing the hexagonal ends of the fibres. ×350.

upon such specimens causes enlargement of the spaces between the hexagons by dissolving the cementing—the interprismatic—substance.

The enamel of human teeth, and, indeed, that of animals, differs in the relative amount of cementing-substance and the number of globules, and, again, in the regularity of the distribution of the two. In one specimen the globules may so predominate that the cementing-substance shows in sections as fine lines, in others the globules may be small, rounded, and surrounded by a relatively large volume of cementing-substance. Again, at different parts of the enamel-rods both arrangements as to relative amounts of the two substances may be seen. It is an apparently constant fact that the cementing-substance is more soluble in dilute acids than is the substance of the calcified globules.

"When enamel deposition proceeds with the utmost regularity striation of the rods is most evident and most regular—*i. e.*, is best marked in perfect specimens of enamel, the striation representing the orderliness with which the layers of globules are deposited" (Williams).

The enamel-rods are crossed at an angle by transverse brown bands, the striæ of Retzius. These are, as compared with the size of globules, broad bands which exhibit parallelism with one another; they are pigmentary deposits. The causes of their presence are not known, but it is probable, from existing evidence, that they represent periodical alterations in the process of enamel-development. They almost follow a series of lines which represent the outer boundary of the enamel-cap at different stages of development; that is, they are most abundant in number in the thickest portions of enamel; least so in the thinnest portions about the necks of the teeth. Noting the mode of formation of the rods, it is evident that they must have a radial direction from the dentin surface outward; the rods are, however, not straight, but pursue an undulating course—*i. e.*, each rod is wavy. In the cusps of teeth, bicuspids and molars, the general direction of the rods is altered, so that they appear to interlace in sections.

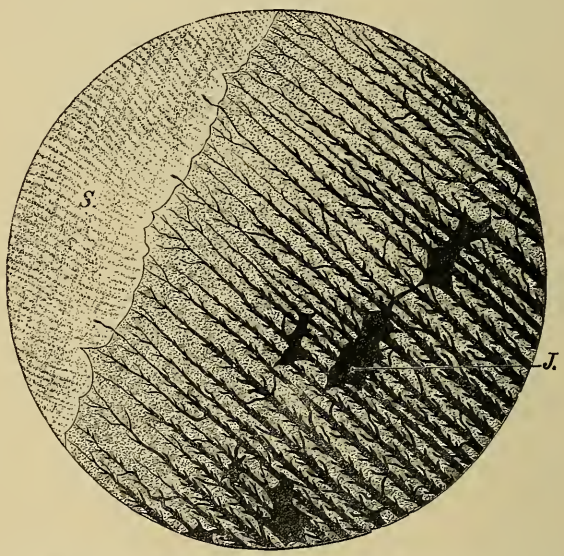
When the crowns of teeth have fully erupted, what remains of the enamel-organ is entirely cut off from its source of nutrition, and in all probability nutritive supply to this tissue ceases prior to the eruption; it is evident, therefore, that any change which occurs in the enamel after the eruption is entirely apart from nutritive influence. The enamel contains at this time all of the materials entering into enamel-composition. It has been asserted in the past, and, indeed, is even believed at the present time, that the enamel undergoes changes of structure with age—grows harder. Cut off from its nutritive source, the only possible way in which change could occur would be in a molecular alteration of the substance of which enamel is formed. Premising that no change whatever occurs in enamel after the disappearance of the enamel-organ at the completion of amelification is an entirely warrantable position, and yet the possibility of such a molecular rear-

rangement as would alter both the physical and the chemical properties of enamel-substance must be conceded. Whether or not enamel does grow harder after its formation is undetermined.

THE DENTIN.

Sections of formed dentin show it to be made up of a calcified basis-substance penetrated throughout its thickness by tubules. The latter exist in such profusion as to occupy in some specimens nearly as much space in the section as is occupied by the basis-substance itself (Fig. 69). In other teeth, presumably those having a less percentage of organic matter, the basis-substance of the dentin predominates. The tubules

FIG. 69.

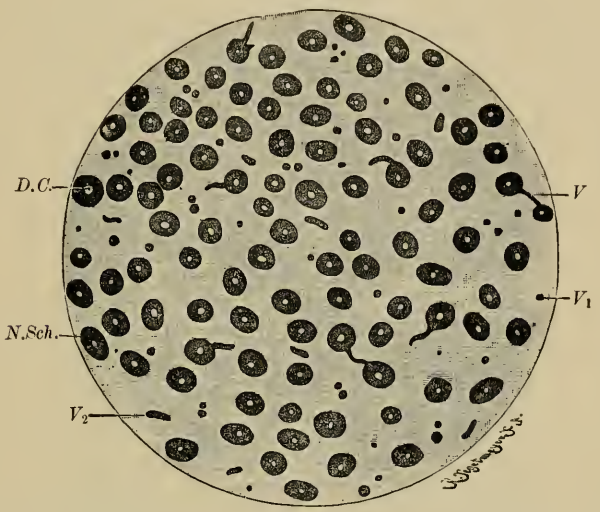


Longitudinal ground-section through the crown of a cuspid of a man æt. 23. Wet ground-section stained by Golgi's method: *J*, interglobular space; *S*, enamel. × 250. (Röse.)

pursue a continuous, wavy, radial course from the periphery of the pulp-chamber to the junction of enamel and dentin. Near their termination at the enamel the tubules divide and subdivide dichotomously. In sections prepared after the Weil method, which makes possible the preparation of laminæ composed of hard and soft tissues and outlines both by means of differential staining, or, in this case, by the Golgi method, it is seen that these dichotomous branches take stain as do the contents of the tubules—*i. e.*, they are the same substance. A transverse section through the dentin exhibits sections of the tubules (Fig. 70). In this specimen saturation of the dentin with Canada balsam preceded the staining, so that the tubules are filled with unstained balsam, showing white. Two distinct substances are seen:

immediately bounding each tubule is a thick layer of substance stained dark, and between these circular areas of staining are more faintly stained areas. If sections be subjected to the action of dilute acids, it is seen that the basis-substance of the dentin is dissolved much more rapidly than the surrounding walls of the dentinal tubuli—*i. e.*, the walls of the dentinal tubuli differ in composition from the basis-substance of the dentin. These walls are called, from their discoverer, the sheaths of Neumann. "If dry and macerated specimens of dentin be subjected to the action of dilute acetic acid (Baumé), the sheaths of

FIG. 70.



Transverse ground-section through the dentinal tubules of the first molar of a child æt. 7: *V.* Koch's and Golgi's methods combined. ×1200. (Röse.)

Neumann around adjoining tubules are seen to be connected with one another by numerous transverse branches."[1] Specimens stained after the Golgi method showed that these transverse connections take stain like the sheaths of Neumann, but that no evidence of the entrance of filaments of Tomes' fibres can be observed, Tomes' fibres being the organic central contents of the dentinal tubule (see later). These transverse branchings are most evident in the most recently formed dentin; least evident in the oldest or peripheral dentin. This virtually marks off the recently formed dentin into defined areas of fully calcified basis-substance, each of which is surrounded by the substance of which Neumann's sheaths and its branches are composed. From its reaction to stains and its behavior toward acids this latter substance is regarded as a transitional material between fully calcified dentin and

[1] Röse, *Dental Cosmos*, 1893.

its organic basis, and a structure quite explicable through the embryology of dentin.

Specimens of dentin subjected to the prolonged action of a 6 per cent. solution of acetic acid do not lose their form, but become pliable, the calcium salts having been removed from the organic matrix. Röse states that if a section of decalcified dentin be placed on a cover-glass and concentrated nitric acid applied, and the slide warmed, the substances between the sheaths of Neumann and their connecting branches melt away; next the connecting branches disappear, and finally the sheaths themselves. In old specimens of dentin there is no evidence of the presence of transverse branches of Neumann's sheaths until sections are subjected to the action of dilute acids, when by the formation of bubbles of carbon dioxid their presence becomes evident. This point is of clinical significance, as it demonstrates that there are in the dentin lines of less resistance to the action of acids.

It has been shown by Hart[1] that the basis-substance of dentin is

Fig. 71.

Main mass of dentin of a temporary tooth, stained with chlorid of gold, decalcified with acetic acid: *F,F*, dentinal fibres, partly vacuoled; *B,B*, basis-substance, traversed by a reticulum. × 1200.

traversed by a fine network of fibres, a connective-tissue stroma in which the calcific process occurs (Fig. 71). Röse[2] regards these as the gelatin-yielding fibres of the dentin.

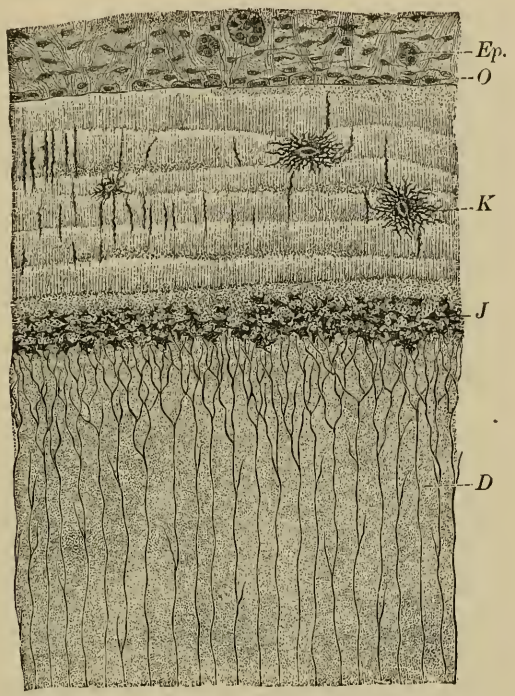
In the border-ground between dentin and enamel and dentin and

[1] *Dental Cosmos*, 1891.

[2] *Dental Cosmos*, 1893.

cementum the dentin usually presents a different histological appearance from the general mass of the dentin. Instead of the orderly subdivision of the dentinal tubules, this portion of the dentin may be occupied by irregular spaces—interglobular spaces. This particular layer of tissue was named by its discoverer, Sir John Tomes, the granular layer (Fig. 72). As he pointed out, the layer is much more marked

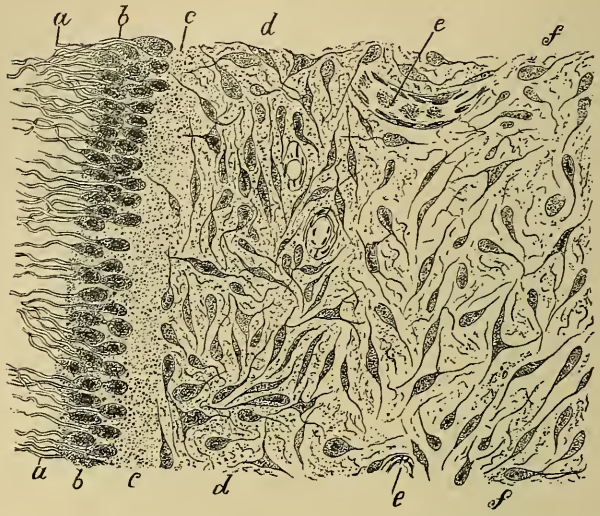
FIG. 72.



Ground-section through the root of a human premolar: *D*, dentin; *K*, cement-corpuscles; *O*, osteoblasts; *Ep*, remains of Hertwig's epithelial sheath; *J*, interglobular spaces. × 200. (Röse.)

beneath the cementum than beneath the dentin. (These spaces will be discussed in Chapter X.) It was first pointed out by the same observer that the organic processes contained in the tubules of the dentin were direct prolongations from the peripheral cells of the dental pulp; that they were processes of the dentin-forming cells—the odontoblasts; these processes are named, from their discoverer, Tomes' fibres. Investigations as to the nature of these fibres have thus far defined them as protoplasmic processes; they are not connective-tissue fibres, and their identity with nerve-fibres is disproved, although their high degree of sensitivity favors such a conclusion. The general direction of the tubules is at the necks of the teeth and in their root-portions at right angles with the axis of the pulp-chamber; in the

FIG. 73.



Margin of dental pulp: *a, a,* dentinal fibrils, pulled out of the dentin; *b, b,* membrana eboris or layer of odontoblasts; *c, c,* transparent zone between the odontoblasts and the cells of the pulp proper; *d, d,* layer of cells closely packed together; *e, e,* bloodvessels; *f, f,* cells less closely placed toward the central portions of the pulp. (Wales' immersion, $\frac{1}{10}$ in. objective.) (Black.)

FIG. 74.

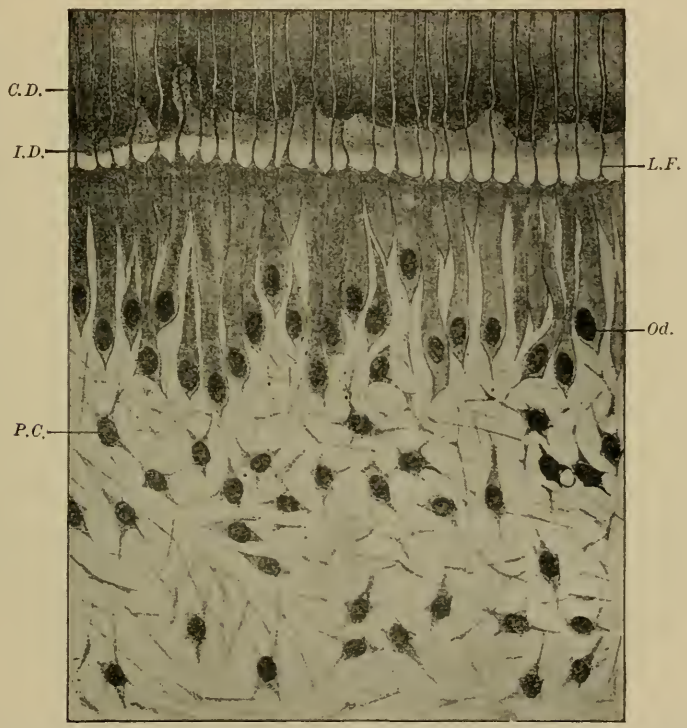
Section of a tooth-pulp: *B.V.*, main bloodvessels of pulp; *C*, origin of capillaries; *N.T.*, main nerve-trunk; *N.F.*, subdivisions of nerve into fibrillæ; *O.D.*, odontoblastic layer; *S.D.*, secondary dentin; *C.G.*, masses of calco-globulin. ×30. (After Röse and Gysi.)

crown portion they proceed radially, the centre of the pulp-chamber being the centre of radiation.

THE PULP.

As foreshadowed in the embryonic tissue of the dentinal papilla, the dental pulp consists of a connective-tissue stroma, an interlacing vascular network, a layer of differentiated formative cells, and a neural

Fig. 75.



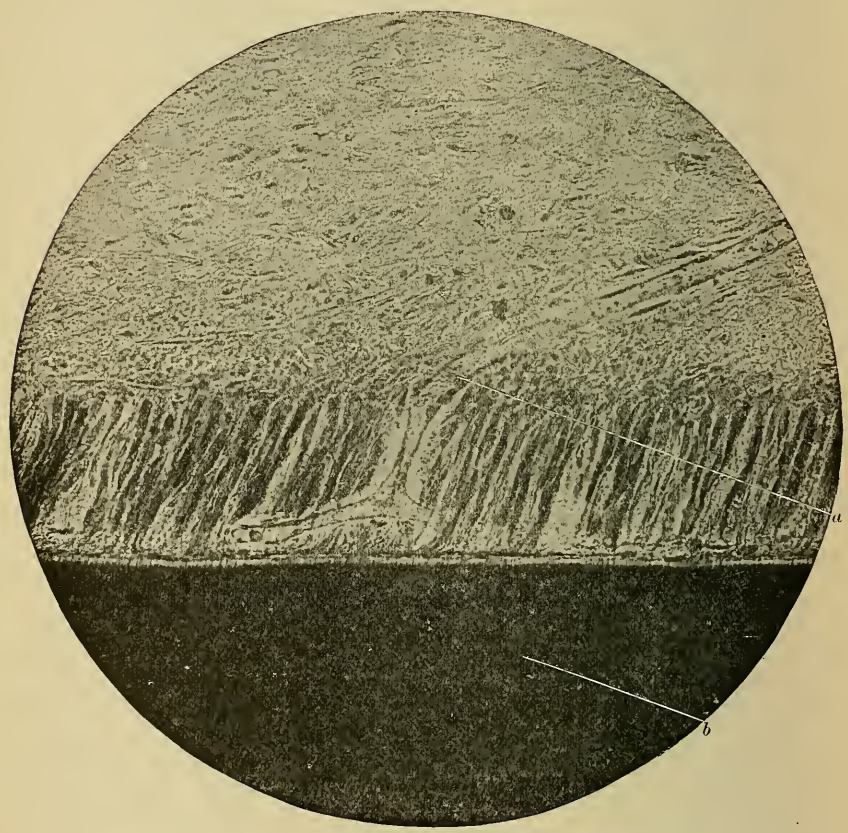
Section of pulp, showing the relations of the odontoblasts to the dentin: *Od.*, odontoblasts; *T.F.*, Tomes' fibres—odontoblastic processes; *I.D.*, uncalcified dentin; *C.D.*, calcified dentin; *P.C.*, pulp-cells. × 800. (Rose and Gysi.)

system. Thus far, no evidences of a lymphatic system have been discovered (Fig. 73).

The general disposition of the tissues of the pulp is as follows: a layer of columnar cells—the odontoblasts—covering its periphery and sending processes—Tomes' fibres—into and throughout the lengths of the dentinal tubule; beneath this layer a closely interlacing plexus of non-medullated nerve-fibres and a capillary network. The middle portion of the pulp is occupied by the trunks of the nerves and by those of the bloodvessels, all of these special tissues being supported by a framework of myxomatous tissue, a modified connective tissue.

If sections of injected and properly stained pulps be viewed under a low magnifying power, it is seen that the arteries and nerves enter the pulp, and the veins leave it by several trunks. Arteries divide and subdivide until they terminate in a capillary network immediately beneath the odontoblastic layer. The nerves are medullated and non-medullated; the former soon lose their medullary sheath, their ter-

FIG. 76.



Section of developing tooth of rat: *a*, bloodvessels entering the odontoblastic layer of cells from the pulp; *b*, dentin. (Williams.)

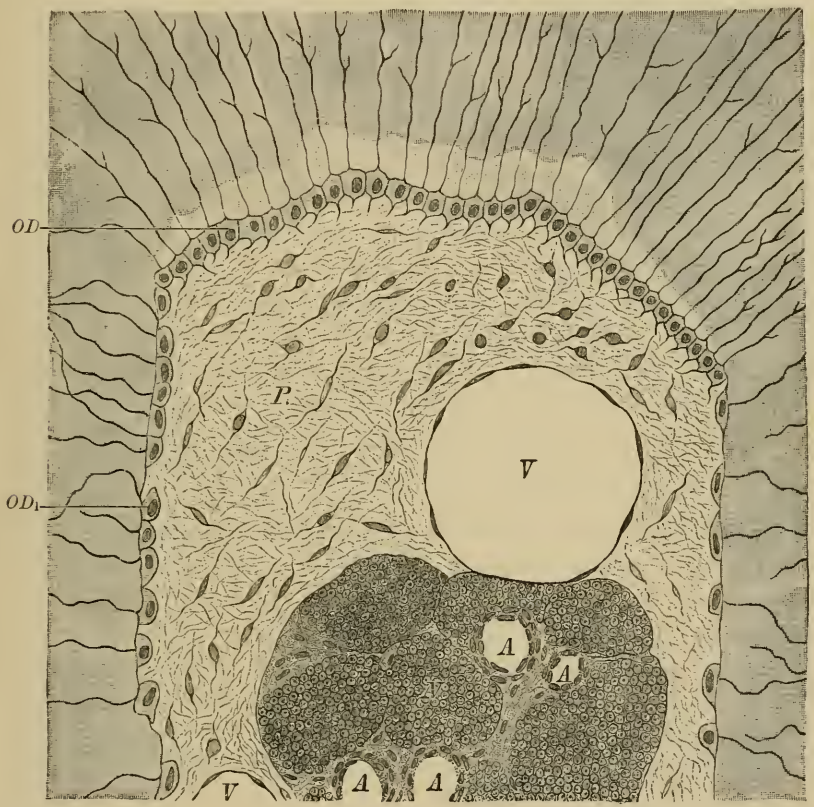
minals apparently ending in a network beneath the odontoblasts. The odontoblasts (Figs. 73 and 75) have the appearance of a thick, cellular boundary membrane. To make out the details of structure of the pulp a magnifying power of 400 diameters must be used.

The Odontoblasts.—The boundary peripheral cells—the odontoblasts—closely resemble columnar epithelium, having between the cells but a very small amount of intercellular substance (Fig. 75). During the period of development the presence of a capillary supply has been detected

in the odontoblastic layer (Fig. 76 and 61).[1] It is to be recalled that, no matter how close their resemblance to epithelial cells, odontoblasts belong to the connective-tissue group. Epithelium never becomes connective tissue, nor can connective tissue ever become epithelium, although the contrary opinion has been held by some histologists. Under abnormal conditions, however, epithelial cells may and do proliferate in the spaces of connective tissue (see Carcinoma).

The odontoblasts are large cylindrical cells which in the pulp of the

Fig. 77.



Pulp from the root of a molar of a man æt. 40. Ground-section after V. Koch's petrifying method: *OD*, active odontoblasts; OD_1, resting odontoblasts, reminding of osteoblasts; *P*, fibrillæ of the pulp; *A*, arteries; *V*, vein; *N*. nerve. Zeiss apochrom. × 250. (Röse.)

young adult are closely massed together, the large nucleus occupying the pulpal extremity of the cell. This gives a pear-shaped appearance to cells whose bodies have shrunk slightly in preparing the pulp for sections. Each cell is seen to send one, and sometimes two, prolongations of its substance into the dentin—Tomes' fibres. Between the

[1] J. L. Williams, *Dental Cosmos*, 1896.

ends of the odontoblasts and the formed dentin a layer of transitional or partially calcified dentin may be seen. At later maturity the odontoblasts appear to have reached their physiological formative-limit —they become smaller (Fig. 77). Under abnormal conditions, however the formative activity may continue until but a faint trace of a pulp-chamber remains.

The Pulp-matrix.—In the child the stroma, the matrix of the pulp, appears to be made up of a loose myxomatous tissue, a structureless basis-substance traversed by bloodvessels and nerves; it is irregularly supplied with large corpuscles, at first round, but which subsequently develop filamentous processes, each cell having two or, it may be, several, giving the cell a stellate appearance—*i. e.*, they are the cells of myxomatous tissue. In the body of the pulp the cells are irregularly arranged, their fibrillar processes forming a loose network. In the root or constricted portion of the pulp the cells appear to exhibit the effects of general compression and have a spindle-like form, and are arranged with their longitudinal axes parallel with the axis of the root. In the mature adult the cells lessen in volume, and the extent and number of their processes increase. Manipulation of such pulps will show that they have increased in toughness. To the naked eye they appear to have become more fibrous. Röse[1] has shown that these fibres do not yield gelatin upon boiling; hence they must be regarded as the transformed processes of cells, and not connective-tissue fibres.

Vascular System.—The arteries of the pulp soon lose almost entirely their muscular coat, and their external coat is reduced to an inconsiderable amount of fibrous connective tissue; the veins remain for an unusual distance without a marked muscular coat. Before the disappearance in the arterial walls and after their reappearance in the walls of the veins the muscular coat is reduced to a layer of extreme thinness, so that histologically the walls of the smaller vessels of the pulp are made up of an endothelial coat, and probably an elastic lamina, an attenuated connective-tissue coat surrounded by the loose stroma-tissue of the pulp. This histological datum has great clinical significance (see Diseases of Pulp).

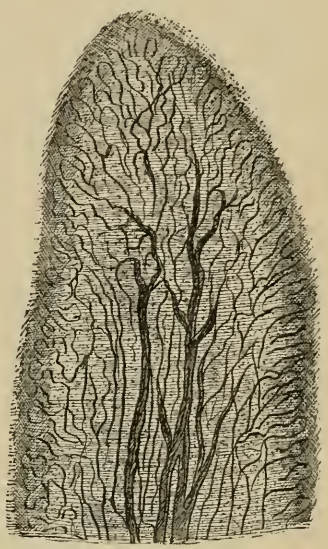
The vascularity of the pulp decreases with age. "In young teeth there are a number of arterial trunks entering the apical foramen, which lessen in number as the passage lessens in size" (Black). Soon after the entrance of the arteries at the apical foramen they divide and subdivide into numberless branches, and finally into a capillary network (Fig. 78), most marked immediately beneath the odontoblasts; the capillaries empty into veinules—these into vein-trunks of relatively large size as compared with the arteries. These vessels pass out of the apical

[1] *Dental Cosmos*, 1893.

foramen parallel with arteries and nerves. This anatomical arrangement of arteries, veins, and nerves, having passage through such a constricted orifice, is of clinical importance.

The Nerves.—The nerves of the pulp, even upon entry to the organ, appear to be both medullated and non-medullated. Whether they belong to both cerebrospinal and sympathetic systems is not made out; nor is their physiological connection with the vessels of the pulp determined. The ultimate bundles of nerve-fibrillæ appear to form an intricate plexus immediately beneath the odontoblasts. By the use of Golgi's stains Retzuis[1] demonstrated that in the mouse the finer nerve-fibres interlace about the bloodvessels, the nerve-trunks themselves following the direction of the bloodvessels. The terminal fibrillæ of the nerves found their way between the odontoblasts, apparently terminating in knob-like extremities between the odontoblasts and the dentin, but showing no evidence of penetration of the dentinal tubuli. No direct anatomical connection has been made out between the nerve-fibres and the odontoblasts, although, as will be shown later, their physiological connection is evident.

FIG. 78.



Point of the pulp of an incisor, injected with Beale's blue to show the bloodvessels. ×25. (Black.)

THE CEMENTUM.

The analogy between dentin and bone is made out with some difficulty; that between bone and cementum is evident—cementum is a modified bone. Examined in gross mass the cementum is seen to be deposited over the entire root and frequently slightly overlapping the extreme edge of the enamel at the neck of the tooth, this being the point at which the first-formed layers of cementum are laid down, the cementum over the apex of the root being that last formed. At the period accepted as the physical termination of tooth-formation the cementum is evenly disposed over the root of a tooth; this is some time subsequent to the eruption of the teeth. Recalling that the development of cementum is a subperiosteal formation of bone, the growth of cementum continues at the sides, and particularly over the ends of the roots, *pari passu* with the decreasing thickness of the peri-

[1] Gustav Retzuis. See Catching's *Compend*, 1896.

cementum, so that at maturity the cementum is disposed as a sheath over the roots of the teeth, thinnest at their necks and thickest over the apices of the roots and also between the roots of bicuspids and molars at their junction. Examined under sufficient magnifying power the cellular elements of the cementum are not so easily made out as are those of bone; but by careful search one may detect the irregular bodies of the cementoblasts, which have formed around them the translucent substance of cementum and remain enclosed in their cement-bed as the cement-corpuscles.

Although there is no decided appearance of lamination in the cementum, there is clearly some evidence of stratification, particularly about half-way down the sides of the roots. In a longitudinal section numerous fine lines may be seen at right angles to the lines of stratification of the cementum; these are former Sharpey's fibres. In the growth of cementum, as of bone, the fibrous elements of the enclosing periosteum have their ends caught in the calcifying bone, and they undergo calcification. These lines represent, therefore, old points and the method of attachment of the pericementum.

It has been noted occasionally that the dentinal tubuli appear to enter the cementum, although their usual mode of termination is in the irregular granular layer of Tomes underlying the cementum. There appears to be a mechanical element determining the regularity of forms of the dental elements; that is, they assume their typical forms under a definite pressure, but when this is variable the histological forms are altered. The first-formed layers of dentin, both in crown and root, it will be recalled, are deposited not against a rigid matrix-wall, but against soft tissue—the inner wall of the enamel-organ, or the epithelial root-sheath of Hertwig; this, no doubt, accounts for the disposition exhibited by the dentin of these parts to assume irregular forms.

THE PERICEMENTUM.

The pericementum is a somewhat complex structure continuous with the general periosteum of the alveolar process; it is also the periosteum lining the inner alveolar walls, is the formative structure of the cementum of the teeth, and, in addition, is the analogue of a ligament, for it serves to bind the root of the tooth to its articular walls—*i. e.*, those of the alveolus. As will be shown later, it is the tactile organ of the tooth—the organ of localization.[1]

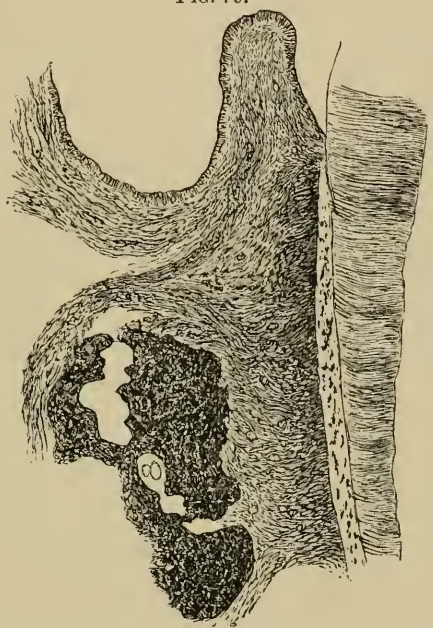
In general terms the pericementum is a fibrovascular, neural, cellular structure, all of whose tissues perform important functions. Viewed in its totality, the structure of the pericementum varies with age; thickest

[1] The description of the pericementum is largely derived from G. V. Black's work upon the *Periosteum and Peridental Membrane.*

when root-formation is just complete, it becomes thinner with age, until in some individuals it is reduced to a lamina of extreme thinness. Its outline-study divides the pericementum into three portions: that adjoining the necks of the teeth, that covering the apices of the roots, and the portion between them.

At the necks of the teeth the pericementum, as stated, is continuous with the periosteum covering the external alveolar walls, continuous as regards its fibrous tissues and also by the bloodvessels and nerves of the part. The neck-edge of the cementum of the tooth (Fig. 79) being at

FIG. 79.



Portion of the side of a root of a tooth, the gum and alveolar dental membrane, and the edge of the bone of the alveolus. A band of fibres is seen passing over the surface of the alveolus and dividing, some passing upward into the gum, others passing more directly across to the cementum. Numerous orifices of vessels cut across transversely are seen between the tooth and the bone. (Tomes.)

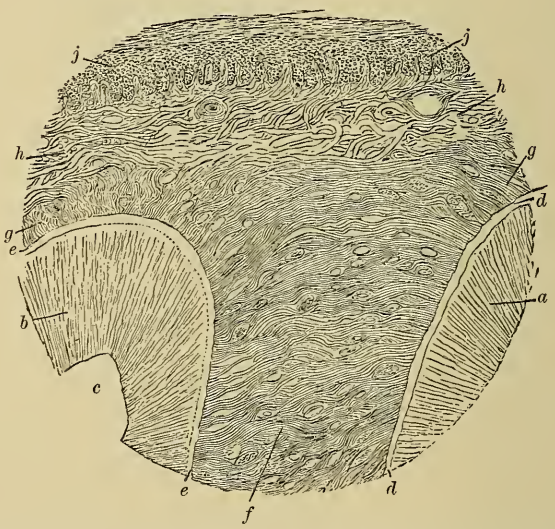
a higher level than the edge of the alveolar process, the pericementum rises to a higher point than the true periosteum. The fibrous tissue of both pericementum and periosteum is continuous with the subepithelial fibrous tissue of the gums. The epithelial tissue of the gums has about the neck of each tooth a rounded margin which leaves between gum and tooth a V-shaped depression. It has been asserted that there are infoldings of the epithelial covering of the gum at this point, forming glands which have a mucoid secretion, the glands of Serres. Black, who termed this epithelial arrangement the gingival organ,[1] later deter-

[1] *American System of Dentistry*, vol. i.

mined that it was without the secretory function attributed to it.[1] As in the skin, the gum is arranged in vascular papillæ beneath the epithelium; these are covered with cuboidal epithelium overlaid by a thick, resistant covering of squamous epithelium.

The Pericementum as a Ligament.—As a ligament joining the cementum to the walls of the alveolus, the pericementum has a definite arrangement of its fibrous elements. That portion of the pericementum extending beyond and upon a level with the periosteum reflected from the outer alveolar walls has, like the periosteum, bundles of fibrous connective tissue ascending toward the crown of the tooth (Fig. 79). This portion of the pericementum unites the pericementum of all of the adjoining teeth together with the alveolar periosteum in one continuous sheet of tissue (Fig. 80). At a higher point within the margins of the

FIG. 80.



Cross-section of the central and lateral incisors below (toward the crowns) the rim of the alveolar wall, or through the necks of the teeth, showing the tissues of the septum and of the gums anteriorly: *a*, portion of central incisor; *b*, lateral incisor; *c*, pulp-chamber of lateral incisor; *d*, *d*, cementum of central incisor; *e*, *e*, cementum of lateral; *f*, fibres of the peridental membrane, extending from tooth to tooth continuously; these are fixed in the cementum of each tooth, and form the tissue of the septum; *g*, *g*, fibres of peridental membrane, which join with the coarse fibrous tissues of the gums; *h*, *h*, epithelial covering of the gums, *j*, *j*. (Black.)

alveolus the fibrous tissue of the pericementum passes horizontally between the root of the tooth and its alveolar wall, the fibres of this portion being the largest and strongest of any part of the ligament. At deeper points the fibres pass from the alveolar walls to points of the cementum farther distant from the alveolar margin, and the tissue is arranged in looser bundles. Around the apical portion of the

[1] *Periosteum and Peridental Membrane.*

root the fibres of the pericementum radiate from the cementum to the alveolar wall. There is no evidence in the mature pericementum of an anatomical division of the tissue into a dental and an alveolar periosteum, although from the mode of formation of the structures and the character of tissue formed by the alveolar cells and the cemental cells such a division might be deemed rational.

The fibres named are of white fibrous connective tissue and their arrangement such that the tooth is swung in its socket. "The oblique fibres protect the tissues of the apical space against the stress of mastication and the horizontal fibres maintain the tooth in position."

The fibres of the pericementum are attached to cementum and to the alveolar walls by large fibrous trunks, which split up into fasciculi of fibres in the body of the pericementum, the fine fibres interlacing with one another. Interspersed throughout the fibrous tissue of the pericementum are spindle-shaped connective-tissue cells—fibroblasts (cells from which fibres develop); they are numerous in the young, almost absent in the aged pericementum.

The other cellular elements of the pericementum are cementoblasts, arranged in an irregular layer against the cementum, the fibrous bundles passing between them for attachment to the pericementum. On the alveolar side osteoblasts are found. Upon both sides multinucleated cells, osteoclasts and odonto- or cementoclasts, are found sparsely at irregular intervals. They are, no doubt, increased in number under some conditions, and at times almost entirely absent.

Black describes peculiar cellular bodies occupying the meshes of the pericementum near the cementum. These have the appearance in section of glandular tissue cut across. The cells appear epithelial in character. The author quoted suggested that these cells and collections of cells belonged to a lymphatic system but now believes them to be epithelial. Considering the mode of formation of the roots of teeth, it is more than possible that they are the remnants of the atrophied epithelial root-sheath of Hertwig.

The Vessels of the Pericementum.—The pericementum is a highly vascular tissue deriving its blood-supply from three sources: one direct, two indirect. The direct supply is from the vessels at the apical space; the indirect, by anastomosis with vessels of the alveolar periosteum which pass over the alveolar rim, and by anastomotic connections with the Haversian system of the bony alveolar walls. The vessels which enter the apical space send branches into the teeth supplying the pulp; other branches pass down the pericementum, surrounded by the fibrous tissue of the membrane, and anastomose freely with the alveolar and the periosteal arteries. The vessels lie nearer to the alveolar than to the cemental walls. The cemental portions of the pericementum are

freely channelled by a plexus of capillaries. The veins pursue the same course.

The Nerves of the Pericementum.—The nerve-supply to the pericementum follows about the same course as that of the arteries. The nerves enter in bundles by way of the apical space; some of them enter the pulp-chamber; others run along the course of the pericementum, dividing and subdividing in their course. Other fibres enter by way of the Haversian canals of the alveolar walls, which split up and at the border of the pericementum form, with fibres from the gum, a fine gingival plexus. Black has observed in some cases the existence of Pacinian corpuscles as nerve-terminals of the pericemental nerves, but believes that the common mode of termination of the fibres is in fine naked filaments.

CHAPTER VIII.

THE SURGICAL ANATOMY OF THE TEETH.

By the surgical anatomy of any part is meant a description of the structure and those relationships of the part which have a bearing direct or remote upon disease of such parts, and a study of those anatomical peculiarities which modify the nature, course, and treatment of such diseases. The surgical anatomy of the teeth, therefore, embraces not only a study of the structure of the teeth themselves, but also a consideration of the environment of the teeth and of all parts with which the teeth have direct anatomical and physiological relationship.

The teeth are to be surveyed from two points of view, mechanical and physiological. Their office being that of the mechanical subdivision of the food, they are to be viewed as instruments built for that purpose. Like other parts of the body, their physical peculiarities and anatomical relations are the immediate factors to be considered in a study of the diseases affecting them. This includes a consideration of all features which are connected with the vital processes and reactions of the body, and a study of their nutrition and innervation, of their relationship with the lymphatic system, and of their associations by both continuity and contiguity.

Beginning at the periphery and proceeding toward the centre several tissues are first to be examined.

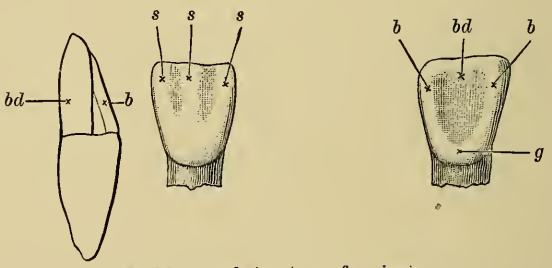
Dental Tissues.

For a short period after their eruption the crowns of the teeth are covered by the structure called Nasmyth's membrane, representing, doubtless, in large part the remnant of the enamel-forming organ. As soon as the teeth are erupted this membrane is deprived of all nutrition, and is therefore a dead tissue—foreign to the body. It is soon worn off the crowns of the teeth by attrition, except in the deep sulci of the bicuspids and molars, and it remains for some time upon the enamel along the gum-margin, where it may later serve as a breeding-ground for micro-organisms.

The tissue subjacent, the enamel, is an entirely extra-nutritional tissue, its source of nutrition disappearing at the termination of enamel-formation. Enamel, once formed, cannot be influenced by any nutritional changes in its substance; it is a dead tissue. It is to be viewed,

first, as a structure designed to do mechanical work; secondly, as to its reactions to its physical and chemical surroundings. Enamel is the hardest and most rigid tissue of the body. "If two blocks of equal size, one of enamel and one of dentin, be subjected to stress, it is seen that the enamel block crushes at a much lower stress than that of dentin, the latter being elastic, the former inelastic; this appears to be true no matter in what axis the enamel is pressed upon; but if a layer of material such as a mat of soft gold be interposed between the enamel and the instrument pressing upon it, its resistance is much increased."[1] If sections of the crowns of teeth be made, it will be seen that there is a mechanical arrangement of the enamel elements and substance fitted to counteract the innate brittleness of the substance itself. First, the enamel surfaces are highly polished, so that there is a minimum of friction between opposing teeth; any increase of roughness or any jaggedness of enamel robs the teeth of this advantage. Secondly, it will be noticed that nearly all of the enamel surfaces which are brought into action during mastication receive mechanical support through an appropriate arrangement of enamel-masses. In the incisor

FIG. 81.



Architectural structure of an incisor.

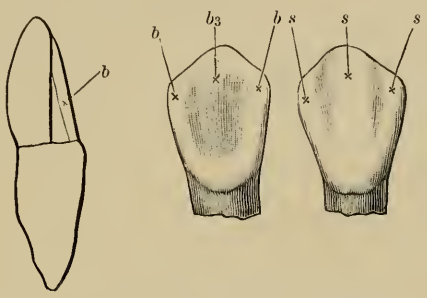
teeth, whose function is the cutting off of defined masses of food, they are primarily blades, which pass one another as the blades of shears, dividing substances which are placed between them. It will be readily seen that in this operation the upper incisors are subjected to greater stress than are the lower. The tendency is to break away the blade of the tooth (Fig. 81, *bd*); this is mechanically guarded against by the presence of two buttresses (*b*, *b*) merging with the angles of the cutting-blade, increasing in thickness as they descend to join a half girdle at the neck of the tooth (*g*). The outer faces of the blade are braced by three stanchions of enamel (*s*, *s*, *s*), one in the middle and one at each lateral edge.

In the cuspid (Fig. 82), which may be regarded primarily as a piercing instrument and secondarily as two cutting-blades set at an angle with one another, the cutting-blades are set with their edges at an obtuse

[1] G. V. Black, *Dental Cosmos*, 1895.

angle, which forms the piercing-point. The edges have each a lateral buttress (*b*, *b*), uniting with a cervical half girdle, and, in addition, a thick buttress (b_3) descends from the point, merging into the girdle. Upon the outer face there are three stanchions (*s*, *s*, *s*), of which the middle one is most marked. In the bicuspid teeth, which, anatomically

FIG. 82.



Architectural structure of a cuspid.

analyzed, are cuspids slightly modified and bound together, as indeed are also the molar teeth, in both molars and bicuspids then, appropriate architectural arrangement of the elements may also be observed. The upper bicuspids are seen to be two cuspids with their cusps or points upon opposite sides. It is seen that these two elements are bound together by two lateral girders or bands which join the lateral girders of the cuspid elements. A similar arrangement is found in the lower bicuspids (Fig. 83, *g*, *g*), although here the double cuspid character of the teeth is not so pronounced as in the upper.

The upper molars (Fig. 84) are structurally composed of three cuspid elements bound together (*g*, *g*), although in the first and second molars a supplementary piece is found, which is weakly girdered to the second of the cuspid elements. It will be observed that this additional piece (*c*), whose junction with the tricuspid arrangement is marked by a sul-

FIG. 83. FIG. 84. FIG. 85.

Architectural elements of bicuspids and molars.

cus, is an element of weakness. In the second molar the additional piece is smaller and not so well attached ; in the third molar it is usually absent.

The lower molars (Fig. 85) are composed of four cuspid elements, the two outer and the two inner being bound together by an anterior and a posterior girder (*g*, *g*). Upon the first and third molars a disto-buccal supplementary piece (*c*) is girdered to the quadrangular mass.

In all of the bicuspids and molars the cuspid character of each cusp

is well maintained in its individual buttresses. The buttresses of the lower incisors and cuspids are much less pronounced than in the upper incisors. The bicuspids and molars, the stress upon whose cuspid elements is mainly lateral, have a buttress and girder arrangement to resist fracture through such a stress.

It is evident that a tooth which has lost any portion of these sustaining structures becomes mechanically weakened in proportion to the extent of the loss. If the lateral girders of a bicuspid or molar be lost through decay, or are cut through, the tendency to fracture of the partially unbound cusps is much increased. If decay in an incisor involve the lateral girders of the tooth, the strength of the cutting-blade becomes correspondingly weakened.

If sections of the several teeth be made and viewed under a low magnifying power, it will be seen that the elements of which enamel is composed have an arrangement admirably fitted to resist the stress to which any particular mass of enamel is subjected. If a mass of enamel be detached from a tooth, and attempts be made to cut it with steel instruments, it will be found that while the hardest steel fails to cut it, a chisel-edge may be made to break away portions of the tissue; this occurs readily only when the tool-edge is applied in the direction of the enamel-rods; if applied at right angles to the enamel-rods, portions are broken away only with much difficulty, and, as a rule, the hardest of tool-steel fails to make any impression upon a perfectly polished enamel surface. Examining prepared sections of entire teeth, it will be seen that the enamel-rods are arranged with their longitudinal axes in such positions that in the impact of mastication stress is received upon the ends of the rods in the lines of greatest resistance.[1]

Defects in the enamel, such as described under malformations of the teeth, have a direct and important surgical bearing. Enamel appears to differ widely as to its physical strength, and also in its degree of solubility in acids. As will be detailed under the head of caries of the enamel, fissures afford opportunities for the lodgement of food-débris, the nidus of lactic fermentation; and, again, the calcic cementing-substance lying between the calcified globules of the enamel-rods is more soluble in lactic acid than are the globules; hence enamel in which cementing-substance is in excess is less resistant.

FIG. 86.

The enamel is thickest and in greatest amount where it receives the greatest stress, shading off until at the neck of the tooth it terminates in a feather-edge, whose outline is that of the gum-margin of the particular tooth, about one-

[1] See, later, Mechanical Arrangement, in this connection.

sixteenth of an inch below which margin the enamel-edge is found, overlaid slightly by the thin edge of the cementum (Fig. 86).

Surgical Anatomy of the Dentin.

Dentin is the second hardest tissue of the body. The texture of this tissue changes, as does that of the other connective tissues, with age. In the young or immature dentin there is a greater ratio of organic matter than in the dentin of a middle-aged person. The increase of calcium salts, the inorganic constituents, has been shown by Black[1] not to be so great as was formerly believed. The average amount of calcium salts in teeth at the age of 11 years is found to be 62.26 per cent.; at 53 years the percentage is 64.56. The average specific gravity at 11 years is 1.066, and at 63 years 2.109. While in the main the increase of specific gravity corresponds with the increase of calcium salts, it is not constant. Between the ages of twenty and forty years there appears to be a cessation in the increase of calcium salts.

Dentin is an elastic substance: a cube of $\frac{8}{100}$ inch side under a stress of 150 pounds is compressed 4 per cent. of its thickness, resuming its form after removal of the pressure. Under a pressure of 238 pounds the cube is crushed. Clear and translucent dentin has a high crushing stress; in opaque specimens it is much lower. An increase in the percentage of calcium salts diminishes the elasticity of dentin; but the amount of crushing stress appears to be governed more by the condition of the organic matrix than by the percentage of calcium salts or the density. When the nutrition of the dentin is interfered with by secondary deposits, or destroyed through death of the dental pulp, the dentin appears to diminish in strength, as seen in the abraded teeth of elderly persons.

The average percentage of organic matter in dentin is 25.36; this diminishes as the density increases. Black believes that the condition of the organic matrix of the teeth has more to do with the strength of the teeth than have the density and specific gravity. The proportion of organic matter, as pointed out by Miller,[2] is not an exact measure of the hardness of the dentin, for many interglobular spaces and wide tubules may account for a high percentage of organic matter, and yet the dentin of the tooth be very dense.

The dentin presents at least three distinct factors for consideration in studying diseases which affect it: first, the basis-material, the calcified matrix of the tissue; secondly, the partially calcified tubes which surround the third factor, the vital protoplasmic filaments of the den-

[1] *Dental Cosmos*, 1895. The statements made relative to the physical properties of dentin are mainly from the same source.

[2] *Micro-organisms of the Human Mouth.*

tin which penetrate its substance and traverse it from the periphery of the pulp to the periphery of the dentin. The calcified basis-substance of dentin is soluble, as is the enamel of the teeth, in dilute acids, such as lactic acid, produced by the fermentation of carbohydrates. When subjected to the action of dilute acids it is changed from the material exhibiting the strength noted above into a mass of about the consistence of cartilage, or softer; deprived of its calcium salts, it still retains its form. Different specimens of dentin exhibit different solubilities. The differences in the percentages of calcium salts and in density do not appear to be sufficient to explain differences of solubility. "The calcium salts appear to be present in the teeth and bony tissues in a combination represented by $(PO_4)_6Ca_{10}CO_3$, or saturated calcium phosphate-carbonate, in a combination which corresponds with apatite, $(PO_4)_6Ca_{10}Fl_2$, phosphate-fluorid of calcium."[1]

It is not known how this material is combined with the basis-substance, the glue-giving substance of the dentin; "whether it is a chemical precipitation, or whether there is a chemical union between the organic and inorganic constituents. In view of the mode of formation of calcoglobulin, the basis of such tissues, it is probable that the latter condition exists. "We should expect to find dentin hard or soft according as this union is firm or unstable."[2]

The general dentin-substance is to be viewed as an interstitial substance, as formed material which in itself necessarily plays a passive part in the disease-processes of dentin. It has been held that the dentin undergoes retrogressive changes, particularly a decrease in the amount of calcium salts in the tissue, not due to the action of extrinsic causes. As a typical example of this alleged metamorphosis are given the changes occurring during pregnancy, when the dentin of the teeth has been said to suffer a resorption of calcium salts; when a more favorable physical condition obtains, a re-deposition of calcic matter is said to occur, restoring the original density of the teeth. Black's analyses indicate that no such resorption and deposition occur. The basis of this belief was in the undoubted fact that during gestation the teeth of the mother suffer markedly from dental caries.

The researches of the same observer showed that there is a greater variation in the density and specific gravity of the individual teeth of a denture than is found in the general average density of many persons.

In line with the same line of reasoning which formulated the hypothesis of resorption and re-deposition of calcic matter should be mentioned the belief of histologists of the Heitzmann school in the possibility of dentin returning to its embryonic form; no sufficient evidence having been adduced in support of such a belief, it must be set

[1] Hoppe-Seyler, quoted by Miller, *Micro-organisms of the Human Mouth.* [2] *Ibid.*

aside, for in addition there are numerous evidences that no such change ever occurs.

Sections of teeth which have been subjected to the prolonged action of dilute acids show that the dentin immediately surrounding the protoplasmic filaments from the odontoblasts is more resistant to the action of the acids than the formed material of the dentin. Noting this comparative insolubility, this portion of the dentin (Neumann's sheaths) has been accepted as a partially calcified tissue. Regarding the general substance of the dentin as a fully calcified tissue and Neumann's sheaths as partially calcified and probably transitional tissue, it becomes a reasonable deduction that any future change in the density of the dentin or any increase in calcic deposits cannot extend beyond the periphery of Neumann's sheaths. This agrees entirely with the condition found in the teeth of the aged, where the dentinal fibrillæ are smaller and the lateral processes of Neumann's sheaths have almost disappeared. New dentin forms then at the expense of the size of the dentinal fibrillæ.

INTERGLOBULAR SPACES.

If many specimens of dentin be examined, it will be noted that in some of them, notably in that portion of dentin underlying the cementum, and less marked in the sub-enamel portion, the general striation produced by the presence of the dentinal tubuli is interrupted and broken into by irregular spaces—the granular layer of Tomes. In some specimens irregularly shaped areas are found widely distributed throughout the dentin, and it will be observed that the dentinal tubuli frequently traverse such spaces, being continuous upon both sides and through them. These are designated interglobular spaces; during life they are filled with soft uncalcified material; they represent areas of non-calcification, apparently an indication that the calcic salts of dentin are deposited in a previously formed soft matrix.

DENTINAL FIBRILLÆ.

Occupying each dentinal tubule, and following its branches, those dichotomous branches beneath the enamel, are processes from the peripheral cells, the odontoblasts of the pulp. Much speculation is still indulged in as to the precise nature of these processes. The most striking feature noted in connection with them is their variable sensitivity. When dentin denuded of enamel is brought in contact with acids, and frequently with sweet substances, a sensitivity is exhibited which varies in degree with individuals. The same result obtains when pressure is brought to bear upon such surfaces, or when applications of heat or cold are made to them. As will be noted later, these cells connected with the pulp, an

organ which has no sense of location, do not localize sensation; the discomfort, pain, or anguish which may be induced by the contact of irritating substances is vaguely located in that branch of the division of the fifth nerve involved. This sensitivity has led to the inference that these protoplasmic processes are in reality nerve-filaments; this one feature supports such a belief; numerous other data contradict it. The processes have been observed as arising directly from the bodies of odontoblasts, but as yet no nerve-fibrils have been detected terminating in the odontoblasts. To regard the odontoblasts as special nerve-terminals would be certainly not irrational, but until nerve-fibrils are discovered ending in the odontoblasts the question is *sub judice*.

Retzuis observes[1] by the use of Golgi's stain that the nerve-fibrils of the pulp appear to penetrate between the odontoblasts and terminate between them and the dentin. The only certain datum is, that these fibrillæ or processes have the power of receiving and transmitting sensation equivalent to, if not identical with, that possessed by nervous tissues. It is certain also that they are portions of the dentin-building cells, and probably possess in some degree the same constructive function.

The Pulp and Its Chambers.

The pulps of the teeth are closely bounded at all parts by dentinal walls; the chamber of lodgement represents, therefore, the precise size and form of the enclosed pulp. The pulp-chambers have forms which correspond quite closely, but not entirely, with the forms of the particular teeth. If sections be made in three diameters of the teeth, these correspondences in form are readily seen, and divergences are observed which are of much surgical interest. Nothing but general rules can be given as to these variations from general types, as the variations may assume very diverse characters (see Figs. 103–113). The pulp is anatomically divided into two parts: that occupying the crown of the tooth is called the body of the pulp, and that occupying the roots the radicular portion or portions. The prolongations from the body of the pulp which correspond with the positions of the cusps are called the horns or cornua of the pulp. It will be observed in many specimens that the cornua of pulps, notably of the upper lateral incisors and upper first bicuspids, very frequently extend as sharp and unduly long projections. It will also be seen by a reference to Figs. 87–102 that the distance from the exterior walls of the teeth to the pulp-chambers is much less at the necks of the teeth than at other parts. This is particularly notable in connection with the upper molars; the distance from the exterior to the pulp-chamber at the cervico-mesio-buccal angle is frequently very

[1] Catching's *Compend*, 1896.

slight. Another peculiarity of tolerable constancy is the nearness of the body of the pulp to the cervico-distal portion of the lower molars.

Fig. 87.

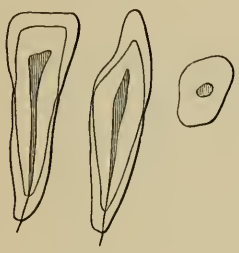


Fig. 88.

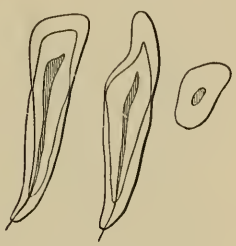


Fig. 89.

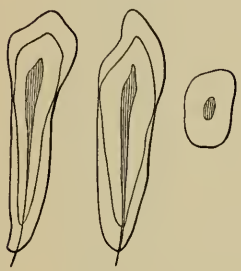


Fig. 90.

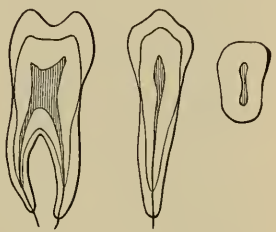


Fig. 91.

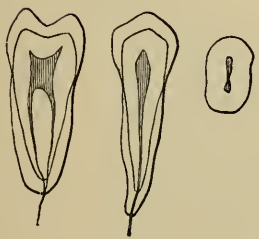


Fig. 92.

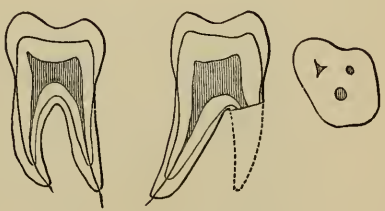


Fig. 93.

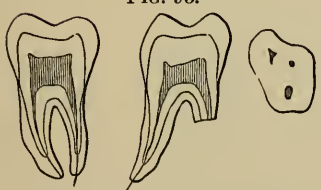
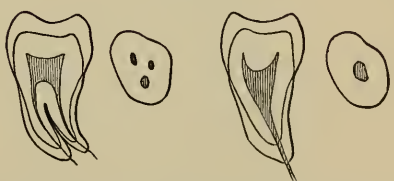


Fig. 94.

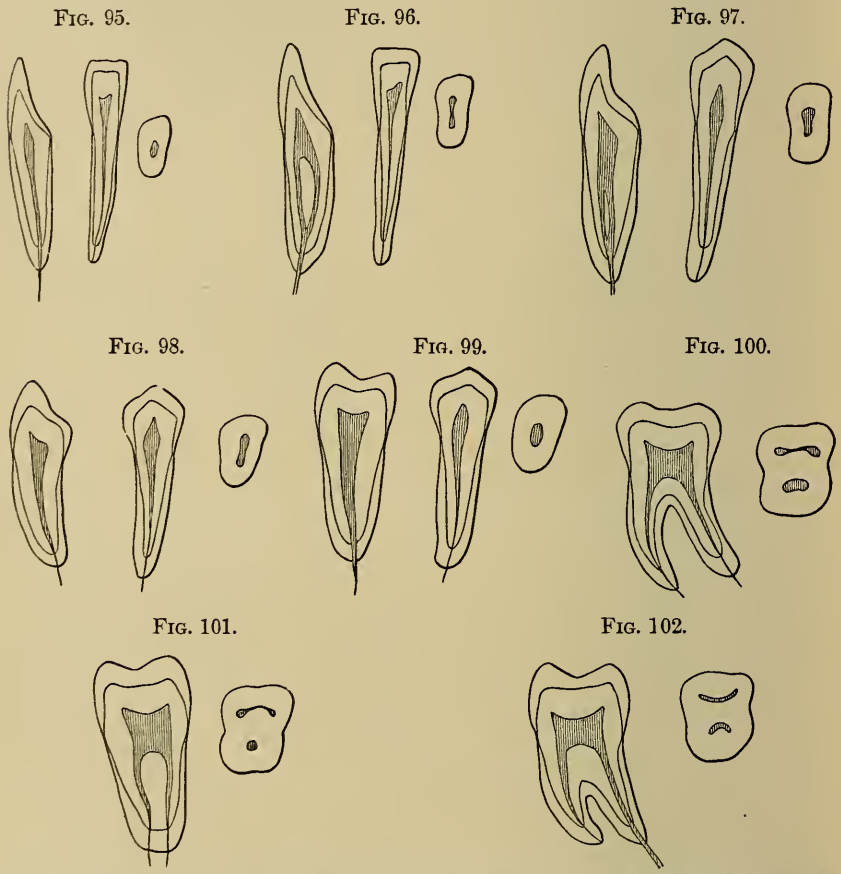


Sections of upper teeth, showing shapes of pulp-chambers and their positions. Figs. 92 and 93, first figures, represent sections through the buccal roots of upper molars; second figures are sections through the palatal root and portion of the anterior buccal roots.

While, as a rule, the external configuration of a tooth is a fair guide to the shape of the pulp-chamber, variations are so common that proximity to the pulp in a carious cavity is more accurately determined by elicit-

ing subjective symptoms than by the objective evidence of the depth of the cavity.

Figs. 103–113 exhibit formalin-gelatin casts of pulp-chambers,[1] showing the great irregularities of form which pulp-chambers may assume, and yet give no external evidence of such irregularities. Figs. 107–110 show the outline-forms of the crown portions or bodies of dental pulps.



Sections of lower teeth, showing shapes of pulp-chambers and their positions. Fig. 101 shows a section through the anterior root of a second molar; an antero-postero-longitudinal section would resemble Fig. 100.

The pulp-chamber decreases in size with age, so that in some cases it is almost obliterated to the end of the root. Secondary deposits (see Secondary Dentin) may fill portions of the cavity and reduce its size in almost any direction. As will be shown, these deposits may occupy the greater bulk of the pulp, and yet the pulp-chamber remain of normal size.

[1] W. H. Richards, *Dental Cosmos*, 1896.

PULP AND CHAMBERS OF TEMPORARY TEETH.

The same rule as to the general size and configuration of the pulp-chamber noted in connection with the permanent teeth holds good in

FIG. 103.

FIG. 104.

the temporary teeth, with this difference, however: it is frequently found that the relative size of the pulp-chambers is greater than in the

permanent teeth, so that exposure of the pulp results soon after caries has invaded the dentin. The radicular portions of the pulp-chambers

FIG. 105.

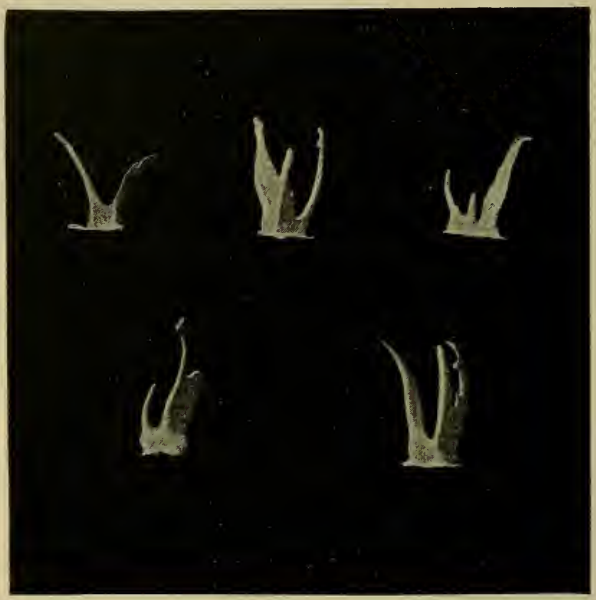


FIG. 106.

of the temporary teeth when fully developed are frequently much attenuated.

The pulp of the tooth contains several factors of surgical interest: first, its vascular, next its neural supply. The pulp is not provided with

FIG. 107.

FIG. 108.

lymphatics, so that effusions which occur in its substance must be taken up by the veins or remain in the stroma of the pulp. The third ele-

ment of interest is found in the layer of odontoblasts, cells which possess a persistent formative and sensory function, and next the

FIG. 109.

FIG. 110.

stroma of the pulp, its framework. The formative cells of the pulp, as will be noted later, are responsive to stimulation, and when stimu-

lated exhibit an exaltation of function expressed in heightened sensitivity and formative activity. These cells, arranged in a layer of toler-

Fig. 111.

Fig. 112.

able regularity, one cell deep, are by mutual pressure forced into an arrangement resembling that of cylindrical epithelium, but having a slight

space between adjoining cells. The odontoblasts appear to abut directly with the dentin, one or more protoplasmic processes passing from each cell into dentinal tubuli. With increasing age the odontoblasts appear to suffer atrophic changes (Fig. 76) (Röse). Beneath the odontoblasts and penetrating the odontoblastic layer for some depth in the developing teeth is a loop-work arrangement of capillaries (Williams).

Fig. 113.

The arterial supply to the pulp enters the apex of the root through several branches, which subdivide and appear to lose their muscular coat at an early period of subdivision. The capillary distribution is, as stated, in a loop arrangement beneath the odontoblastic layer. The capillaries join veins of very large size, in which the middle coat appears to be absent for a long distance. It is evident, then, that should pulp arteries and veins lacking this muscular coat be subjected to increased intravascular tension, danger of injury to their walls is imminent, and occurs in hyperæmic and inflammatory conditions.

The nerves, entering by way of the apical foramen, are both medullated and non-medullated; in the peripheral distribution all of the fibres are non-medullated. Whether the non-medullated fibres are purely vasomotor, and the medullated the sensory fibres, is not determined. A point of much surgical interest relates with the sensory function of the pulp nerves. As emphasized by Black,[1] the pulp is a truly internal organ, and, like many other internal organs, it has no sense of location

[1] *American System of Dentistry*, vol. i.

—*i. e.*, it is not the touch-organ of the tooth, so that when subjected to irritation these nerves react as do the nerves of other typical internal organs—reflect the sensation to some other branch of the same nerve. The classical illustration of this phenomenon is the pain of certain hip-joint diseases: the pain is not felt at the disease-seat, but upon the inner side of the knee; the pains of hepatic disorders are referred to the right shoulder-blade; those of inflammation of the eye-curtain, the iris, to the brow.[1] This is a fact of great clinical importance in dentistry, viz., pains due to disorders of the pulp are not accurately located, but are referred to distant points. The pulp nerves are, however, singularly intolerant of pressure, responding in painful paroxysms to any pressure, internal or external.

The stroma of the pulp has a distinct surgical interest. It is to be recalled that originally the pulp consisted of a mass of undifferentiated mesoblastic cells in a structureless basis-substance. Later, some of these cells become differentiated into dentin-forming cells; the dentin is finally deposited in a finely fibrillated stroma of fine gelatin-yielding fibres. At maturity the cells of the pulp are of a myxomatous type, contained in an apparently structureless matrix; of polygonal form in the crown of the pulp, these cells show a semblance to a fibrillated arrangement in the radicular portion of the pulp. It has been shown by Röse[2] that these cells and their processes are not ordinary connective-tissue cells; they do not yield gelatin upon boiling. As the pulp increases with age the bodies of the cells grow smaller and their fibrillar processes become more marked, until in an old tooth an appearance of fibrous tissue is seen. The matrix-cells are of pathological interest from the fact that they appear, under some conditions, to assume the formative office of odontoblasts, deposits of dentin occurring in the pulp-substance. The changes which occur with age in the tissue of the pulp may be likened to sclerosis.

Cementum and Pericementum.

At the apex of the root the arterial and neural supply to the interior of the tooth is continuous with the same structures to the external vital structures of the tooth, viz., the pericementum and the cementum.

The pericementum is the touch-organ of the tooth, the nerves of which possess the sense of location. The pericementum is the mutual periosteum of the cementum of the tooth and of the enclosing alveolar wall; reflected over the external alveolar wall this periosteum becomes continuous with the general maxillary periosteum. In addition, the pericementum is the ligament binding the tooth in its articular (the alveolar) walls. As a periosteum it is a source of nutrition to the

[1] Black, *Ibid.*

[2] *Dental Cosmos*, vol. xxxv.

cementum and to portions of the alveolar walls, so that interference with its vascular supply is followed by malnutrition of these tissues, the effects being governed by the extent of the interference (see General Pathology). As a ligamentous tissue, its fibres, as shown by Black,[1] have a peculiar arrangement. The bundles of fibrous connective tissue which pass from the alveolar walls to the cementum are oblique in their general direction, the fibres passing from a point nearer the margin of the alveolus to a deeper portion of the root.

The nerves of the pericementum are accustomed to a degree of pressure represented in the amount of force necessary to crush the particles of food; if subjected to a greater stress, they rebel.[2] Similarly, if their functional activity is exalted in hyperæmic disturbances, they become cognizant of very slight pressures and react more strongly. It is to be remembered, however, that the teeth rarely receive direct stress, the movements of the teeth in mastication being more of a rotary and laterally moving character, than perpendicular.

The elasticity of the pericementum is to be regarded also as an adjuvant to the local circulation; by its movements the blood is pumped through the vessels of the part. This fact becomes important when it is recognized that in some dentures increasing age is accompanied by a lessening of the volume and elasticity of the pericementum.

The power of recovery of the pericementum after injury appears to be very great. It will be observed that the pericementum has two sources of vascularity: one from the apical vessels, that from which the vascular supply of the pulp arises; the other, an anastomotic circulation from the alveolar walls, directly and indirectly from the general alveolar periosteum. When the apical vessel-trunks have been obliterated as the result of disease the pericementum receives from the anastomotic circulation a blood-supply practically sufficient.

The cementum maintains its vitality so long as the pericementum is intact; in the condition just mentioned it is evident that the apical portion of the cementum dies, or is at best very ill nourished. The layer of cementoblasts (osteogenetic cells) retain their function so long as the pericementum is intact, and under varied conditions exert their constructive function in an irregular manner (see Hypercementosis).

Surgical Relations of the Teeth.

The several groups of teeth—incisors, cuspids, bicuspids, and molars—all have surgical relationships differing in each jaw and in each dental group.

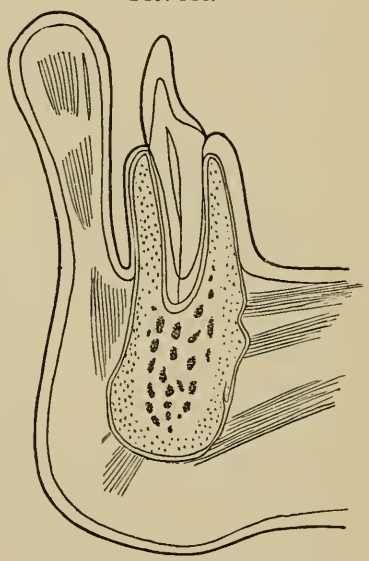
[1] *Periosteum and Peridental Membrane.* [2] Black, *Dental Cosmos*, 1895.

THE LOWER DENTURE.

Incisors.—The lower central incisors, as well as the lateral incisors and cuspids, receive their main vascular supply from the continuation of the inferior dental artery, the passageway of which is not so well marked a channel as in the portion of the jaw posterior to the mental foramen. The neural supply is from the inferior dental nerve, which returns through the mental foramen recurrent branches, which pass to the muscles about the mouth. This association of nerves is made clinically evident in many cases where dental irritation of these teeth causes painful symptoms referred to the soft parts about the mouth.

Direct surgical interest as to the lower incisors associates with the relations of the apices of the roots of these teeth with their bony surroundings. A section through the symphysis of the jaw will show the bone to be very dense at the site of the section. Sections passing through the axes of the central incisors will show that beneath the apices of the roots is a mass of irregularly channelled cancellated bone, bounded by an inner and an outer layer of cortical bone, the inner or lingual layer being much more dense than the outer (Fig. 114). As a rule, the nearest point of approach of the root of the tooth to the surface is upon the outer or labial side, where but a thin layer of cortical bone may overlie the apex of the root in the incisor fossa. On the lateral incisors the layer of bone is usually a little thicker, and thicker yet over the apex of the root of the cuspid, provided the root be not abnormally short. In all of these teeth, and, as will be shown later, in all of the lower teeth, the minimum thickness of dense cortical bone is not over the apices of the roots, as in the upper jaw, but at points about midway between the necks of the teeth and the root-apices. This will explain why abscess upon the roots of the lower teeth rarely discharge over the apices of the roots of the teeth.

FIG. 114.



A longitudinal section through a lower central incisor and its neighboring parts.

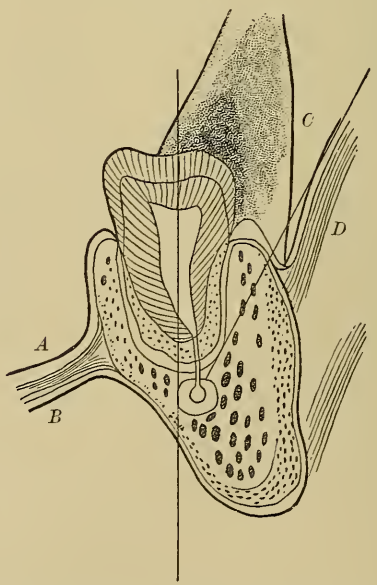
Occasionally the roots of the lower incisors are overlaid labially by an unusually dense layer of cortical bone; this fact, taken in connection with the observation that at the submental portion of the jaw,

immediately beneath these roots, is a relatively thin layer of cortical bone, furnishes the solution as to why an abscess upon these teeth may open beneath the chin.

Bicuspids.—The bone overlying the roots of the bicuspids at their lingual aspects is sometimes relatively thin, as it forms the wall of the sublingual fossæ. Upon the labial face the cortical bone is in greater amount, although thin. The spaces between the first and second bicuspids usually marks a site immediately above the mental foramen, although the opening may be posterior, or in some cases anterior, to the position named. If the roots of the bicuspids are abnormally long, they may encroach upon the area of the foramen. This occurs most frequently with the root of the second bicuspid, affections of which tooth may cause diffused pain, apparently owing to the proximity of the root-apex to the nerve-trunk at the foramen.

Molars.—In studying the surgical and anatomical relations of the lower molars, the anatomical subdivision of the lower jaw becomes most apparent from every point of view. It is anatomically and clinically divided into an alveolar portion and the maxillary portion proper, the

FIG. 115.



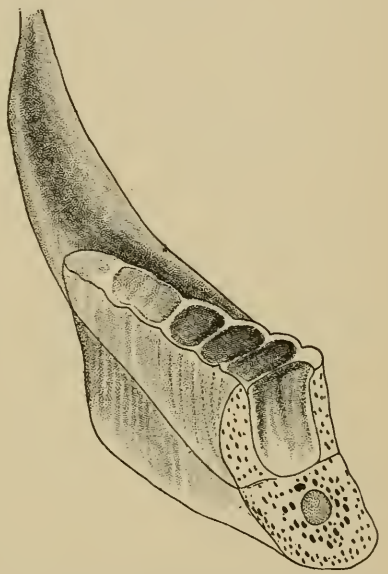
Showing the relations of the roots of the lower third molar with the cavities of the mouth and neck, and with the external bony wall: *A*, cavity of mouth separated from *B*, the cavity of the neck, by the mylohyoid muscles; *C*, base of the coronoid process; *D*, muscles of the cheek.

alveolar portion—that portion in which the teeth are embedded and which surrounds them—being set upon and inside the body of the bone. The fact that the alveolar portion is set inside the body division

is not so apparent in the anterior portion of the jaw, although it will be readily seen that the dental arch has a smaller radius than the maxillary arch; but at its extreme posterior portion the relationship is most evident, where the outer edge of the coronoid process is some distance external to the buccal faces of the lower third molar (Figs. 115 and 116). It is with this superimposed and inserted mass of bone that dental diseases are almost entirely concerned. According to Allen,[1] "all diseases affecting this portion of the bone are to be regarded as dental."

The boundaries of the molar portion of the alveolar bone are to be clearly kept in mind (Fig. 116). If sawn away from the body of the bone, it will have the form of an irregular quadrangular pyramid, the base of which represents the section between the first molar and the second bicuspid, one side the alveolar margins about the molars, another the buccal portion of the bone adjoining the external oblique line, its inner side, the alveolar portion, having the internal oblique line (mylohyoid ridge) as a margin; its remaining side, the bone, overlying the inferior dental canal. These four sides merge into a point beyond the inner and outer divisions of the base of the coronoid process. In position the pyramid lying upon one side, that forming the roof of the inferior dental canal, is seen to have gradually altering anatomical relations advancing toward the apex of the pyramid. Viewed upon the inside, one edge of the pyramid lies along the boundary line, the internal oblique line, the line of insertion of the mylohyoid muscle which divides the cavity of the mouth from the cavity of the neck; with its covering of mucous membrane the muscle forms the greater portion of the floor of the mouth. As this line approaches the surface of the mouth progressively to immediately back of the third molar, its relations, and therefore the relations of the cavity of the neck to the roots of the teeth, also change. So that while the roots of the first molar are rarely deeper than the mylohyoid ridge, the second and more frequently the third molar roots may pass to a greater depth (Fig. 117). It will also be observed that

FIG. 116.

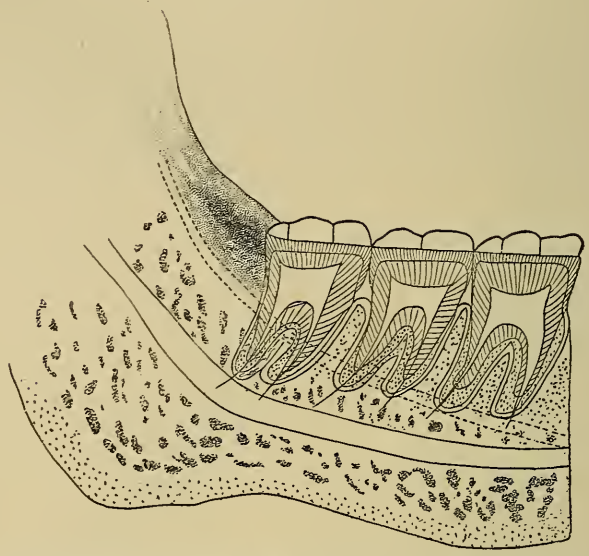


Showing the relative position of the posterior portion of the alveolar bone (the dentale) with the maxilla proper.

[1] Harrison Allen, *Human Anatomy.*

the smaller size of the alveolar arch, as compared with the mandibular arch, causes the molar portion of the alveolar bone to project bodily into the cavity of the mouth, so that a vertical line passed through the

FIG. 117.



Section of the molar teeth and corresponding portion of the inferior maxilla; the dotted line represents the position of the mylohyold ridge, the open space beneath the inferior dental canal.

vertical axis of the third molar would fall directly into the cavity of the neck without passing through the body of the bone (see Fig. 115). This relationship of parts makes the thinnest portion of bone overlying the roots of molars at the apex of the distal root of the third molar. A drill passed through the frequently thin covering of cortical bone at this point emerges almost exactly in the groove lodging the mylohyoid branches of the inferior maxillary nerves and arteries.

The root may terminate at a point some distance behind the anterior pillar of the fauces. Upon the external or buccal face of the bone it will be seen that the position of the third molar is at times some distance posterior to the outer branch of the coronoid process, the continuation of the external oblique line, so that a greater distance separates the roots of the second and third molars from the external surface of the bone than with any of the teeth of a denture. Abscess upon the roots of particularly the lower third molar, therefore, finds the path of least resistance as to pus-exit, first, by destroying the pericementum of the tooth and finding exit at the gum-margin, or, if the outer alveolar plate is not entirely walled in by the base of the coronoid process, through the alveolar process near the gum-margin (Fig. 115). An abscess may, by penetrating the lingual alveolar wall, open far back

in the mouth, if the roots of the tooth are not deeper than the mylohyoid ridge; if the roots do penetrate beyond this ridge, it may open in the neck in the submaxillary triangle.

In some cases the roots of the second and third molars may immediately overlie the inferior dental canal, instead of being slightly inward, and the tissue intervening between the apices of the molar roots and the canal may consist of the very thin layer of perforated cortical bone which forms the roof of the canal. In some cases a molar root may be so deeply embedded as to encroach upon the canal, lessening its lumen and causing more or less compression of the inferior dental vessels and nerves. Pressure from such sources is, no doubt, the cause of obstinate maxillary neuralgias, which would be greatly exaggerated in disease-conditions about the pericementum, accompanied by inflammation or even hyperæmia. In impacted third molars the pressure of some part of the tooth may cause great distortion of the course of the canal (see Chapter X.). The anatomical relations of the third lower molars are such that apical abscess upon them will have tardy vent, or else open in unusual situation.

The blood-supply to the inferior maxilla through the inferior dental artery—large single trunks which traverse the bone longitudinally upon both sides—may be seriously impeded or checked by pressure upon the trunk as it enters or shortly after its entry to the canal, and thus, necrosis of half the maxilla is a probable danger. In many cases, however, where the inferior dental vessels have been obliterated upon one side necrosis does not occur, the anastomosis of the facial artery with the dental about the mental foramen continuing sufficient circulation to maintain vitality.

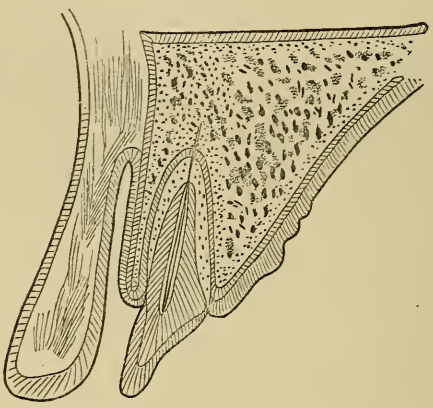
THE UPPER DENTURE.

As in the lower jaw, the line of the symphysis, a thickened plane of bone, is an impediment to the extension of disease from either side. The demarcation between alveolar and maxillary portions is as clearly defined as in the lower jaw. In general terms, at the period of maturity a plane passing through the floor of the antrum and beneath the floor of the nose divides the alveolar from the maxillary bone. As a rule, this plane would also rest upon about the height of the maxillary vault. In general character and in both vascular and neural supplies the upper jaw presents features differing from those in the lower jaw.

The alveolar bone is formed, as in the lower jaw, of two layers of cortical bone, between which lies cancellated bone, chambered by the several alveoli for the roots of the teeth. Of these two plates, the outer is the thinner, and, unlike the outer alveolar boundary of the

lower jaw, is of nearly uniform thickness over the roots of all the teeth (Fig. 118). As in the lower jaw, a greater thickness of bone

FIG. 118.



Section of an upper incisor tooth, its attachments, and anatomical relations.

overlies the roots at their lingual aspects. Differences as to the depth of the alveolar process are more apparent in the upper than in the lower jaw, measured, first, by the height of the dental vault, and, next, by the distance from the occlusal edges of the teeth to the reflection of the mucous membrane from the cheek to the gums. The apices of the roots of the several teeth in the upper as well as in the lower jaw are usually about one-eighth of an inch or more above the line of reflection of the mucous membrane. The common vascular trunk from which the upper teeth derive their arterial supply is a short branch that divides into an alveolar portion which enters the posterior wall of the antrum; running in the muco-periosteum of that sinus, it subdivides into numerous branches, some of which enter the alveolar portion of the bone as posterior dental arteries supplying the molar and bicuspid teeth. The posterior buccal portion of the alveolar walls, with the overlying gum-tissue, are supplied from the same source. The palatal portion of the alveolar process is supplied by a separate arterial trunk, the descending palatine, which emerges upon the palate at the posterior palatine foramen, running forward thence to anastomose with the terminal branches of another small division of the internal maxillary artery, the naso-palatine, at the incisive foramen. The anterior teeth receive their vascular supply from the second branch of the common arterial trunk, viz., the infra-orbital artery. As this artery runs in the infra-orbital canal, it gives off descending branches which penetrate the antrum; running under its muco-periosteum, it supplies the anterior portion of the sinus and branches penetrate the bone supplying the anterior teeth, anastomosing freely with the posterior dental arteries.

Disease, effusions, etc., may occlude the external alveolar arterial supply and cause necrosis of the dependent plate of bone, and yet the vascular supply of the internal alveolar plate remains intact. Again, the external alveolar bone overlying the anterior teeth may suffer occlusion of its circulation without affecting the posterior external alveolar blood-supply. This will account for the frequently limited character of necrosis of the upper alveolar bone, as compared with necrosis occurring in the lower jaw, and will serve to explain the forms of sequestra in certain cases.

The neural supply to the upper teeth, derived from the superior maxillary division of the trigeminus, corresponds almost exactly with their arterial supply. The complex character of the anastomoses of the dental nerves will explain in some measure the varied character and indefinite location of pain of dental source in the upper jaw.

Central Incisors.—The surgical relations of the roots of the central incisors are associated with the floor of the nose. Recognizing the bony floor of the nose as a common surface, with the plane of bone forming the hard palate, and the teeth-bearing bone as an arch-shaped rampart set upon this plane, it will be seen in the majority of cases that the roots of the central incisors do not extend deeper than the base of the alveolar bone: but in rare cases their roots may impinge upon the bony floor of the nose, and a very thin layer composed of peri-cementum, bone, and muco-periosteum, may separate the apices of the roots from the floor of the nose.

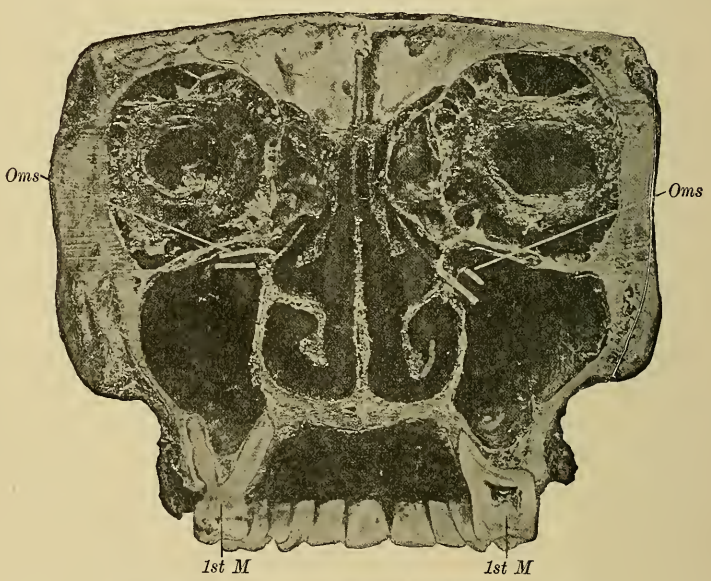
In some cases the outer alveolar plate overlying the apices of the roots may be very dense and comparatively thick; and the palatal alveolar plate be thin, and furnish the line of least resistance to pus-collections. This may also be true of the lateral incisor; the principal surgical interest of which, however, lies in the common and abrupt curvature of its root-apex, usually pointing away from the median line. It is of interest to note that this tooth is occasionally not found. Whether the lack of development arises from no cord ever being given off for it, or whether the tooth-follicle at some very early stage suffered atrophic changes, is not precisely known; it is probable, however, that no follicle has been formed.

Cuspids.—The surgical interest as to the cuspid teeth is, first, in their very deep implantation, in many cases their roots extending beyond the nasal floor, but to its outward side; and, next, in the unusually compact character of the bone overlying their roots. Very frequently the bone overlying the apex of the root is of extreme thinness.

Bicuspids.—The first bicuspid is chiefly notable for its two roots, labial and lingual. When these roots are divergent, which as a rule are

convergent, the palatal root may be overlaid by only a thin layer of tissues. As with the molars, disease may attack but one root alone. With the second bicuspid an additional anatomical relation of surgical interest may be found. The root of this tooth may underlie the floor of the anterior portion of the maxillary sinus, although this relationship is not so common as with the next tooth in the dental series. It is to be remembered, as shown by Cryer,[1] that the cavity of the antrum becomes larger with age; and while in the young adult the roots of all of the posterior teeth may be separated from the floor of the antrum by a considerable thickness of bone, progressive resorption with the consequent enlargement of the antral cavity may carry the floor of the latter not only down to, but in some cases beyond the apices of the roots of the teeth (Fig. 119).

Fig. 119.



Oms. opening maxillary sinus; *1st M*, first molar. (Cryer.)

It will be observed that the true maxilla begins at the situation of the first molar, to extend outward far beyond the outer alveolar wall as an enormous process, the malar process, which is but a shell of bone, forming the greater portion of the outer wall of the antrum. While with the first molar usually only the palatal and disto-buccal roots underlie the antrum, the projection of the malar process carries the boundary of the antrum outwardly beyond all of the roots of the second and third molars. Upon lifting the lip of the living subject the

[1] "Studies of the Maxillary Bones," *Dental Cosmos*, Jan., 1896.

projection of the malar process is evident, its anterior edge overlying the first molar tooth. The line of the floor of the antrum usually lies about a quarter of an inch above the reflection of the mucous membrane from the cheek to the gum, and if a sharp drill be passed backward, upward, and inward from the reflection line above any of the molars, the antrum is readily penetrated. It will be seen that the anatomical relations of the roots of the molar teeth to the floor of the antrum, particularly in mature and elderly persons, are such that disease of the apical pericementum is necessarily followed by involvement of the tissues of the floor of the antrum. Moreover, extraction of teeth in some cases would be inevitably accompanied by fracture of the bony floor.

The apex of the palatal root of the second molar lies in close proximity to the groove at the palatal base of the alveolar process, which accommodates the posterior palatine artery, so that slipping of an elevator in extraction might cause injury to this artery. The tuberosity of the palatal process which lodges the roots of the third molar is a loosely cancellated knob of bone, having but weak boundary walls. At the base of its palatal portion lies the posterior palatine foramen. The frail boundary walls have been broken in attempts at extraction of this tooth, producing fracture of the condyloid process. Occasionally the excavation of the antrum extends far into this tuberosity, and forcible attempts at extraction of a conical molar have resulted in driving the tooth bodily into the antrum.

The Teeth as Mechanical Appliances.

Having studied the physiological aspects of the teeth, they are to be surveyed as mechanical implements in which derangements of the primary mechanical design are followed by morbid alteration in dental physiology.

The teeth, as mechanical instruments, are divided into four well-recognized anatomical classes: incisors, cuspids, bicuspids, and molars; each group, and indeed each member of each group, is of mechanical design and position for a definite office. According to the form and position of a tooth, it is fitted to receive and resist stress applied to it. Stress in excess of this amount, or received from an abnormal direction, is to be regarded as a menace to the integrity of the tooth. The particular pathological interest in an excess or irregularity of stress lies in the response of the vital parts of the tooth implicated. The tissue or structure mainly concerned in the vital reactions of the teeth toward mechanical overwork, and, contrawise, toward disuse of the teeth, is the pericementum.

The effects of overuse and of disuse will be discussed in detail in the section dealing with diseases of the pericementum.

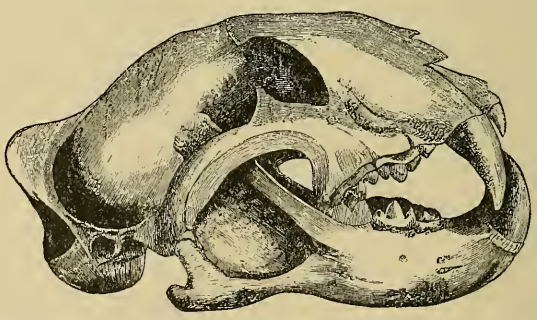
THE UPPER DENTURE.

Incisors.—The broad cutting-blade of the central incisors receives a stress which tends to force it directly outward. In the normal denture this stress ceases as soon as the posterior teeth are brought into occlusion. The length of time the stress is in operation depends upon the extent to which the upper teeth overlap the lower; if the overbite be great, the stress is proportionately increased. The roots of these teeth have as a mechanical provision against the stress a sectional form resembling a scalene triangle having much rounded angles, whose base is parallel with the edge of the cutting-blade, offering a broad surface of resistance. The tendency of the force of mastication, incision in this case, tending to drive the teeth away from the median line, the base of the triangle is placed at an angle with a plane transverse and at a right angle with the symphysis (Fig. 121). In the lateral incisors the same provision is observed as to the arrangement of the base of the section triangle of the root, which is decidedly more scalene (Fig. 122).

While it would be apparently more in harmony with anatomical design that the length of the root should correspond with the amount of stress involved, so that teeth having a long overbite should have long roots, no such rule is observable, for it is frequently seen that teeth having broad and long cutting-blades have very short roots; but, as a rule, the broad and long cutting-blade is associated with a root section triangle having a broad base.

The Cuspids.—To appreciate the mechanical design of the cuspid

FIG. 120.

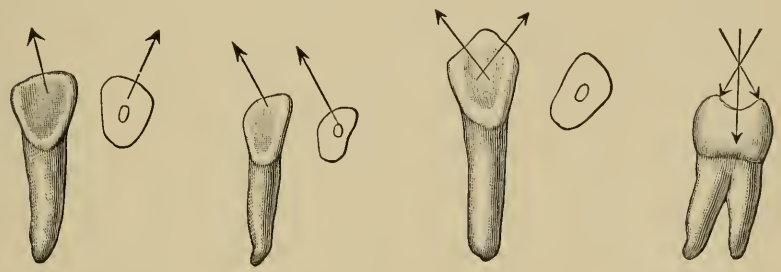


Side view of the cranium of a tiger, with the mouth slightly opened to show the relative position of the great canines. (Tomes.)

tooth, it must be studied as it occurs in its typical form, as the canine tooth of the carnivora. In these animals the enormously developed canine is formed to serve as a weapon of defence and offence, being provided with a very extensive and deep implantation to insure strength and firmness; this characteristic is a constant associate of the true canine. In the preda-

tory carnivora the upper and lower canines are arranged in such manner that the lower, the mandibular canines, lock in front of the upper canines, so that any substance caught between them is firmly held, and it is necessary to tear the held substance to free it. Bonwill has pointed out another function for these teeth: it will be observed, when the jaws of an animal such as a lion or tiger are wide apart, that the tips of the canines upper and lower are in contact with one another, the lower in front and outside of the upper, thus the lower jaw is doubly held,

Fig. 121. Fig. 122. Fig. 123. Fig. 124.



in guides (Fig. 120), posteriorly by the temporo-maxillary articulation, anteriorly by the cuspids on both sides. In closing, the jaws can move in but one position—that which will bring the posterior carnivoral teeth exactly in the relation of shear-blades; the canines, therefore, guide the other teeth of a denture into their proper occlusion. They markedly limit the lateral or rotary movement of the jaws; so that in the human denture or in

Fig. 125. Fig. 126.

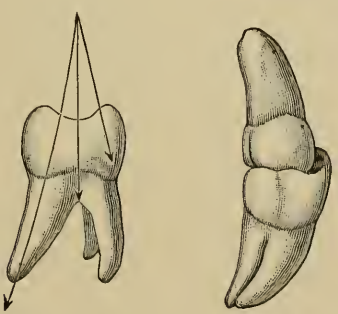


Fig. 127.

animals where the rotary movements of mastication are pronounced, the canine becomes an insignificant tooth—it is reduced to an incisor. The tip of the canine cusp in man is occasionally so long in those dentures having a long overbite that its office as a guide to correct occlusion is marked, and in such cases the prominent canine eminence overlying the length of the cuspid root is correspondingly large. In all typical human canines or cuspids the presence of the double cutting-blades at an angle with one another is the characteristic feature; as both blades are functional, the tendency of the root of this tooth is to

be forced directly outward (Fig. 123), the direction of the resultant of the two forces received by the blades; the stress is resisted by the unusual root length and by its broadly rounded outer, labial wall, and again by the density of the overlying bone.

The Bicuspids.—Mechanically the human premolars (the bicuspids) are double cuspids. Reverting to the function of the preceding classes of teeth, it will be seen that the incisors incise and cut off definitely sized masses of food, and on examining the nature of the occlusion of the upper cuspid and its antagonists it will be seen that the surfaces are so disposed as to flatten the mass previously incised, to press it between four surfaces. The double cuspid and the triple cuspids behind them —the upper molars—are so formed that the cutting-blades of the outer or buccal sections are sharper than those of the inner, the palatal, or lingual sections, and the cusps of each diminish in depth progressively to the last molar. While the cutting-edges of the molar-cusps are fitted for cutting, it is the squeezing surfaces which are of greatest functional importance. The stress upon the roots of these teeth is in three directions: outward, when the buccal cusps are in action; inward, when the lingual cusps are in action, and there is a constant vertical stress (Fig. 124). The roots of these teeth are, therefore, of elliptical section to resist the inward and outward strains; and cylindro-conical to resist the upward stress. These facts are of direct clinical importance in the restoration of lost crowns by fillings or artificial crowns, for if the occlusal surfaces are not so arranged that the root receives stress in all of these directions, and all stresses are not properly balanced, overstrain or disuse of some portion of the pericementum follows. This applies also to the incisor teeth, on which broad surfaces are sometimes added to artificial crowns without due regard to the distribution of stress in other parts of the denture.

The first upper bicuspid, having deeper cutting-blades and a broader occlusal surface, requires a corresponding increase in the amount of root resistance afforded; this is found in the usual bifurcation of the roots of these teeth.

The Molars.—While the molars receive stress laterally upon their outer and inner cusp-sections, the greatest stress to which they are exposed is nearly vertical. The stress outward is a very strong one, as affecting the roots of the teeth, mainly because of the increased breadth of the tooth. Pressure upon the buccal edges operates at a greater distance from the axes of the teeth than in the bicuspids: hence the stress is greater. This fact is of clinical importance in that fillings and artificial crowns are frequently made with too great a cusp length, thus increasing the stress upon the buccal roots. It will be seen that when the palatal cusps of the molars are in use the stress received by them is

usually in the axis of the palatal root, and the tendency toward displacement is minimized (Fig. 125).

The upper third molar when in use presents a cusp-arrangement interesting when the nature of the occlusion is studied. The usually single palatal occludes with the broad depression between the cusps of the lower third molar (Fig. 126). The mechanical arrangement—a dull conical wedge in opposition to a broad basin—is one fitted for the crushing of hard but frangible bodies which rest in the basin of the lower molar, and are pressed upon by the wedge of the upper molar. A given amount of muscular power operates at this point with the greatest force, because, as will be seen, the lower jaw is a lever of the third class, the alveolar bone forming its long arm, so that the further back a tooth is in the dental series the greater will be the stress with a given amount of muscular power.

The tripod disposition of the roots of the molar teeth is in correspondence with the stress directions upon their crowns; the rocking action caused by the outward, inward, forward, and backward stresses is fully met by such a disposition of resistances. These movements, greatest in the first molars, find in this tooth a greater root-divergence. With a lessening of the lateral stresses in the hinder teeth there is a lessened root-divergence (Fig. 127), until with the third molar, where the stress is mainly vertical the roots are closely placed together, and the stress of direct pressure is diffused by a curving backward of the roots.

In the foregoing, as in the succeeding, remarks it is fully recognized that during the period of the development of the teeth and jaws and during the eruption of the teeth influences are at work which may explain many of the anatomical peculiarities mentioned and cause variations from them, yet these features as described are in the main characteristic of the several teeth.

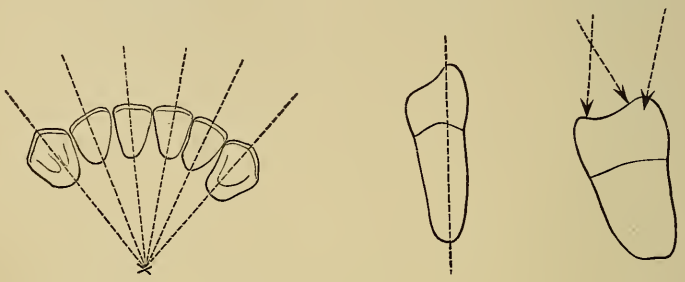
THE LOWER DENTURE.

Incisors.—The stress upon the lower incisors tends to force them inward, a displacement resisted not alone by the angles at which the elliptical section roots are set, but the pressure tends to close, to drive together the teeth of the lower incisive arch which afford mutual support to one another (Fig. 128). The broader cutting-blade of the lateral incisor is accompanied by an increase in the size of the supporting root. The posterior cutting-blade of the lower cuspid is the longer, and it is upon this blade or upon its outer face that the greatest stress is received. Support against this stress is afforded, first, by the incisive arch, and, secondly, by the curvation of the deeply implanted root.

Bicuspids.—The lower bicuspids are not truly bicuspid, the first being a cuspid with a partially formed second cusp. It has been de-

monstrated by Bonwill that there is a well-defined anatomical reason why this tooth should not be formed as the upper bicuspid—*i. e.*, the inner cusp would be a constant interference with the cusps of the occluding teeth. It will be noted that the inward inclination which the buccal wall of this tooth has in common with the buccal walls of all the lower posterior teeth brings the axis of the cusp nearly in line with the axis of

Fig. 128. Fig. 129. Fig. 130.



the root (Fig. 129), hence the tendency to lateral displacement is neutralized; the pressure received upon the lingual surface of the bicuspid is antagonized by the angle at which the axis of the tooth is set to the perpendicular. The eight anterior lower teeth form an arch against which the anterior segment of the tongue exercises pressure. In a normal denture the tendency to outward displacement which would arise in consequence of this pressure is prevented by the upper dental arch mechanically holding the lower in position. When the teeth of the upper arch are lost, this muscular pressure is frequently sufficient to cause outward displacement of the lower incisors. This tendency is more pronounced when from any cause the attachment of the lower incisors is lessened.

The crown of the second lower bicuspid, having the inner cusp as an edge rather than a true cuspid section, has its greatest stress received upon the buccal cusp. The crown of this tooth is bent inward, so that its buccal cusp lies in a line which passes through the axis of the root. The stress is in two main directions, inward and outward; although, as in all of the posterior teeth, there is also the antero-posterior stress. In all of the lower teeth in normal occlusion there is a tendency to mechanical displacement in a forward direction, which becomes usually marked when the loss of a tooth deprives the tooth posterior to it of its anterior support.

Molars.—In the dental segment represented by the second bicuspid and the three molars Bonwill has pointed out that the buccal cusps are rounded and dull, while the lingual cusps are the sharp segments, an arrangement the reverse of that found in the corresponding upper

dental segment. The greater extent of pressure upon these teeth is received upon the buccal segments, and the tendency to displacement is therefore inward or lingually.

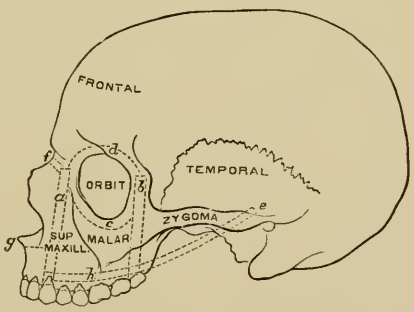
The Jaws and Denture as a Mechanical Apparatus.

Recognizing, as has been shown, that disarrangement of the mechanical disposition of any tooth is followed by an alteration of the local physiology, the principle needs elaboration from another aspect—that is, the teeth are to be viewed not only singly, but as integral parts of the entire dental system; which is, first, the sum of the single teeth, together with the mechanism and physiology of the motor apparatus. These are to be studied: the jaws themselves and associated parts, and the details of their function.

As emphasized by Harrison Allen,[1] the maxillary bones, particularly the superior maxillæ, although in close relationship with the cranial bones enclosing the great nervous ganglia, yet belong to the visceral skeleton;[2] they are but attachments to the neural skeleton.

The teeth are arranged and are contained in recesses of two portions of this visceral skeleton, one, a fixed or immovable mass of bone, which has a definite distribution of its masses fitted to afford great resistance and to dissipate force, to minimize the amount of shock transmitted to those portions of the neural skeleton, the basal cranial bones, which

Fig. 131.



Architectural scheme of the upper jaw: *a*, nasal column; *b*, malar column; *c*, infra-orbital arch; *d*, supra-orbital arch; *e*, pterygoid column; *f*, upper nasal half-arch; *g*, lower nasal half-arch; *h*, maxillary arch.

enclose the brain-masses. The second, a movable mass, operating from centres which are in apposition with the brain-case proper, and in which another mechanical arrangement dissipates energy transmitted to the areas of contact between the visceral and neural skeleton.

A. H. Thompson,[3] enlarging upon Allen, divides the disposition of

[1] "Studies of the Facial Region," *Dental Cosmos*, 1874.

[2] See also Hertwig's *Embryology*.

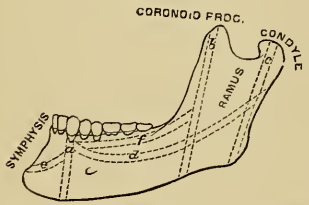
[3] *Dental Cosmos*, 1893.

the bone-masses of the superior maxilla (Fig. 131) into columns affording resistance to the force received. A nasal column, its axis passing through the cuspid tooth, and ascending to the inner margin of the orbit, crossing the nasal bone at an angle to meet a corresponding extension from its fellow of the opposite side. A second column rising perpendicularly from the margins meets the orbital rim at its outer side; the rim of the orbit furnishes two arches, a supra- and an infra-orbital arch. A third column or girder passes in a curved line nearly at right angles with the other columns. "The latter column serves to brace the bone against the lateral stress of mastication; the other columns against that of direct occlusion." "The loss of the cuspids deprives the face of its normal fulness due to the canine column, a fulness not replaceable by mechanical means." Minor braces may be outlined, all defending the maxilla against injury from the forces received by it.

The greatest force of impact received by the molar teeth is by the superior maxillary articulations, dissipated through arches and projections of the neural skeleton, so that concussion of the cranial contents is reduced to its lowest limits.

Examining the detached inferior maxilla, it will be seen that it is also provided with strengthening columns (Fig. 132), which protect it from injury by the stress received upon it. The mental column, as shown, is a line of great resistance, but behind it the point of emergence of the mental vessels and nerves, the mental foramen, is a line of weakness, protected, however, by the usually increased thickness of the cortical bone on this line. Despite this, however, a perpendicular line passing through the mental foramen represents usually the line of least resistance.

Fig. 132.



Architectural scheme of the lower jaw: *a*, mental column; *b*, coronoid column; *c*, condyloid column; *d*, body arch or curve; *e*, arch of the symphysis; *f*, molar arch.

The lateral columns of the external and internal oblique lines are the columns of great resistance against both lateral and direct stresses. The condyloid column is an efficient brace at the rear edge. It is found with some degree of frequency that these columns are traversed by lines of lessened resistance. A constant line of lessened resistance crosses the coronoid and condyloid columns transversely and at an angle on a line above the attachment of the internal pterygoid muscle.

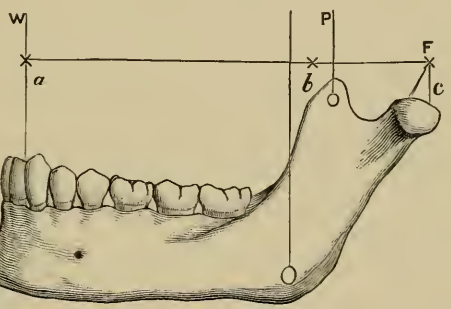
As tersely stated by Thompson, the superior maxilla with its teeth represents the static element of mastication; the inferior maxilla with its teeth and its attachments, the dynamic element.

The lower jaw is a lever of the third class: the condyle is at the fulcrum; the horizontal distance from the condyle to a line passing through the coronoid process and the angle of the bone, the short or power-arm; from the condyle to the mental column is the length of the long or weight-arm. The power of the lever is represented by the action of the temporal, masseter, and of the pterygoid muscles, particularly the internal, when right and left muscles act synchronously. The weight is the amount of pressure brought to bear upon substances between the teeth. The weight upon the incisors, these teeth being at the extremity of the weight-arm, is less in amount than that upon teeth posterior to the incisors; with a given force of muscular contraction, the greatest power is exercised at the third molars, in accordance with the mechanical law of

$$W \times L = P \times L' \text{ or } P \times bc = W \times ca.$$ (See Fig. 133.)

Black[1] records the amount of force which can be exerted by the jaws. He found that the maximum force of those whose jaw-power

Fig. 133.



was tested, to be 175 pounds on incisors, and in the same individual 240 pounds on the molars. The minimum force recorded for adults was 30 pounds on incisors and 70 pounds on molars. While in many of the cases tested the ratio between the force exerted through the incisors approximated one-half that exerted through the molars, which corresponds quite closely with the mechanical positions occupied by incisors and molars on the mandibular lever, many other cases showed a ratio of 1 to 3, and some 1 to $1\frac{1}{4}$. These discrepancies occur with sufficient frequency to show that the condition of the pericementum of teeth has more to do with the force exerted upon them than has mechanical position. With an average distance from condyle centre to the cutting-edges of the incisors of four inches, and a distance from condyle centre to the axis of muscular action of about $1\frac{1}{2}$ inches, when a force of 100

[1] *Dental Cosmos*, June, 1895.

pounds is brought to bear upon the incisors the muscles are exerting a contractile force of $266\frac{2}{3}$ pounds.

$$4 \times 100 = 1\tfrac{1}{2} \times x = 1\tfrac{1}{2}\ x = 400 \quad x = 266\tfrac{2}{3} \text{ pounds.}$$

The amount of force necessary to crush food-stuffs by direct action ranges from 30 pounds for tender meats to 90 pounds for tough, fried meat; hard crusts resist a pressure of 250 pounds; hard candy requires in the neighborhood of 100 pounds. The stress exerted upon temporary molars, about 70 pounds, is quite sufficient to crush ordinary meats. In affections of the pericementum of a hyperæmic nature the sensitivity of the pericemental nerves is much increased, so that less pressure can be exerted than in health. According to the investigator whose work is quoted, many persons involuntarily fail to exert sufficient masticatory power owing to the debility of the pericementum of one or more teeth.

The Dental Mechanism.

The dental apparatus operating upon an arm of a lever and against a fixed base has, normally, a definite arrangement of its members, making one of the most admirable anatomical structures conceivable. Dentistry is indebted to W. G. A. Bonwill, of Philadelphia, for the working out of the details of the dental mechanism.[1]

The teeth are arranged in two arches, an upper and a lower, the upper being the larger and overlapping the lower at its labial and buccal aspects. In the lingual cavity the lower arch projects beyond the upper. These arches are usually parabolic, although at times semi-elliptical, and in some cases the dental series, upper and lower, form three sides of a trapezoid having rounded angles. As a rule, the distance from the centre of one condyle to the mesio-occlusal angles of the lower central incisors is about four inches, which is also the distance from the centre of the right condyle to that of the left, lines of junction through these points forming an equilateral triangle.[2] It follows, of course, that in a normal denture the equilateral triangle is also to be included in the lines joining the centres of the glenoid fossæ and running to the junction of the upper central incisors.

The occlusal surfaces of the teeth are arranged in a definite manner. The first bicuspid tooth of the upper jaw is the anatomical basis from

[1] This description of the dental mechanism is largely derived from the written works and personal communications of Bonwill.

[2] In Bonwill's written communication he attaches an occult significance to a series of measurements, based upon the equilateral triangle in the measurement of the sizes of the teeth and the determination of their positions. The facts cited are in the main correct, but are not, as Bonwill believes, a conclusive argument against the truth of organic evolution. His laws are as all anatomical laws subject to variation, although unusually constant.

which deductions may be drawn: first, the deeper the cusps of this tooth; for the character and depth of the cusps of all the teeth may be determined by an examination of those of the first bicuspid, the more the upper teeth overlap the lower, the overlapping being most marked in the incisors, and least with the third molars; secondly, the teeth of a denture have not their cusp-edges on a plane; a plane surface laid upon teeth is in contact with the edges of the central incisors, usually with the cuspids, bicuspids, and the anterior cusp of the first molars: the cusps of the second molar are at a distance from the plane, and those of the third molar at a greater distance—that is, the line of the molar cusps curves upward (Fig. 134). If the cusps of the teeth be very long, the

FIG. 134.

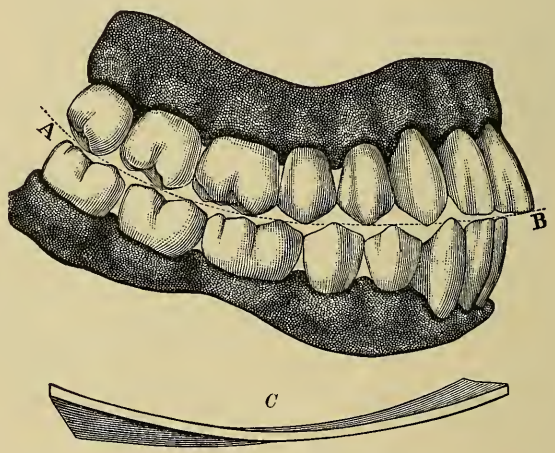
Front and side views of the teeth and jaws.

cuspids may sink beneath the surface of a plane resting upon bicuspids and incisors. The longer the cusps the more pronounced is the upward curve. A plane laid upon the lower teeth touches the occlusal edges of the incisors, cuspids, and only the distal cusps of the third molar; the second bicuspid, first and second molars, are below the plane, the first molar farthest below. The depth of this depressed curve is, as in the upper teeth, governed by the length of the cusps. In cases where the cusps are abraded the occlusal surfaces are reduced almost but not entirely to a plane. Thirdly, it will be observed that the buccal cusps of the teeth, from the second bicuspid backward, are at higher points than the palatal cusps, so that if a curved and plastic plane be pressed so that its surface is in contact with the cusps of all of the teeth of one side it will assume the form shown in Fig. 135. The longer the cusps of the teeth—*i. e.*, the greater the overbite, and in consequence the greater the upward curve of the arch—the more the surface of the plane will be bent.

Placing the two dental arches in normal relation with one another, it is seen that each tooth (Fig. 134) is in occlusal contact with two teeth of the opposite jaw, with the exception of the lower central incisors and the upper third molars, which teeth have but one antagonist. The interspace between any two teeth of either jaw passes nearly through the vertical axis of a tooth in the opposite jaw. The arrangement is such that should a tooth be lost, its occluding teeth still have each an antagonist, except in the case of the four teeth which have but a single antagonist. Teeth having but a single antagonist are lost through a process of gradual extrusion after the loss of their antagonist.

In the act of incising food the jaws are first separated and the lower jaw advanced until the cutting-edges of the upper and lower incisors

FIG. 135.



The horizon of the line of occlusion and plane of occlusion.

are opposite one another. While the jaws are in this position with the incisors in occlusal contact they do not bear alone the stress of whatever muscular force is applied, but it will be seen that the distal cusps of the third molars, the highest points of the lower dental arch, advance and meet the distal cusps of the upper second molars, higher points of the dental arch, so that when the incisors are in edge-to-edge occlusion, although all of the other teeth are separated to an extent governed by the overbite, the dental arch is supported posteriorly by contact of the last molars, thus preventing undue stress upon the incisors. As the incisors glide past one another the lower jaw recedes, and an increasing molar area comes in contact.

The teeth of man, being of the omnivorous type, adapted both for incising and grinding food, the lateral movement of the jaws is of extreme interest. While the posterior teeth of man, notably in those

individuals having long cusps to their teeth, have a distinctly carnivorous character, yet it is of secondary prominence as compared with the herbivorous character of the molars.

To do the actual work of the mechanical subdivision of food, of course the cusps and molar surfaces of the teeth must be in effective contact. After a mass of food has been excised by the incisors and pressed between the cuspids it is passed to the posterior teeth, and the lower jaw is swung toward that side, when it is seen that from cuspid to molar the rounded buccal cusps of the lower teeth rise until they are in contact with the sharp buccal cusps of the upper teeth. Upon the opposite side the teeth are in contact at two or more points, supporting the arches against overstrain, but are not in functional use; only one side of the jaw is in functional use at one time, the food-masses being shifted from side to side until mastication is completed.

The mechanism for the proper subdivision of the food, the function of mastication, being as described, it is evident that aberrations of tooth-arrangement, the loss of a single tooth or of a column of teeth, will be followed by a corresponding interference with mastication. Aside from the æsthetics offended by the abnormal positions of teeth, there is a distinct pathological interest involved in them, as shown in the chapters on Malpositions of the Teeth and Diseases of the Pericementum.

CHAPTER IX.

DENTITION: ITS PROGRESS, VARIATIONS, AND ATTENDANT DISORDERS.

The process of teething, the eruption of the teeth, technically called the process of dentition, may be defined as the vital operations through which the partially formed teeth are elevated from their beds in the maxillæ until the gum is pierced and the crowns assume their anatomical positions in the dental arch. It includes a consideration of the completion of root-formation and the coincident development of the alveolar walls. It is evident, therefore, that a study of the process of dentition is a continuation to the study of dental embryology; being, in fact, the later stages of a developmental process which began in the sixth week of embryonic life in an involution of the epithelium upon the summits of the jaws, and has its physiological termination in the apical constriction of pulp-tissue due to the formation of the anatomical apex of the root. Physiological dentition is divided into two groups: first, the eruption of the temporary or deciduous teeth—first dentition; secondly, the eruption of the permanent teeth—second dentition.

Causes of Eruption.

Explanations as to why teeth erupt are unsatisfactory and many of them conflicting; none which has been advanced is sufficient to explain all of the observed phenomena. Nearly all of the hypothetical explanations advanced may be included in two classes—the dynamic and the vital. Those who subscribe to the dynamic view hold that the process of dentition is due to the pressure of the forming root upon the tissues beneath it, causing reciprocal pressure on the overlying tissues; resorption of the latter tissues occurs and the tooth-crown emerges through the gum. The vital theory regards eruption as a physiological process which carries the developing teeth, practically uninfluenced by the pressure-element, into their positions in the dental arch. The principal objections stated to the dynamic theory[1] are that stunted teeth erupt; that is, teeth having roots so short that downward pressure is insufficient to explain their emergence in the dental arch are found in the dental arch; and, also, that the distance travelled by the crown of

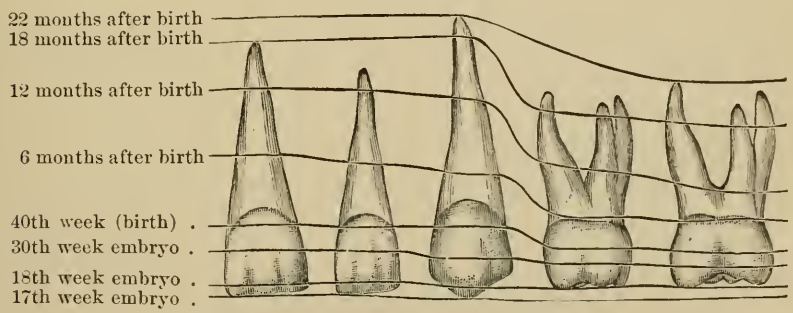
[1] Tomes, *Dental Anatomy*, fourth ed.

a tooth in the process of eruption is greater than the length of the root. These objections are, however, more apparent than real; and comparing the observed phenomena with the dynamic theory, it is probable that the pressure of the developing root, and the tissues about it, are at least very important elements in the process; and yet it must be borne in mind that the changes occurring in the entire mass of alveolar bone are as marked as is the alteration in the position of a tooth in its eruption. The alveolar changes do not cease until long after eruption.

Process of Dentition.

Dentition may be said to begin when the crown of the tooth approaches the covering of the bony crypt in which it is lodged and resorption of the edges begins. This occurs shortly after the time of beginning root-formation. The relations of the parts at this period are as follows: each tooth-follicle is lying in a crypt of bone, separated from the bony walls by thick layers of fibro-vascular tissue upon all sides. The bone of the floor of the crypts is the roof of the inferior dental canal in the lower jaw. In the upper jaw the floors of the crypts are not far beneath the level of the infra-orbital foramen, from which point they will be far distant in the mature jaw. The crypts are almost roofed by the bone overlying crowns, although a fissure exists in the roof of each crypt at its anterior portion. The teeth when in position will have their necks somewhat beyond the site of these fissures; their roots, still unformed, will extend from this point to nearly the base of the existing crypt, and instead of lying loosely in their roughly spherical crypts, the future roots will be embraced by deposits of bone separated from the roots by an attenuated portion of the follicular wall. It will be seen, therefore, that not only must a larger deposit, in point of

Fig. 136.

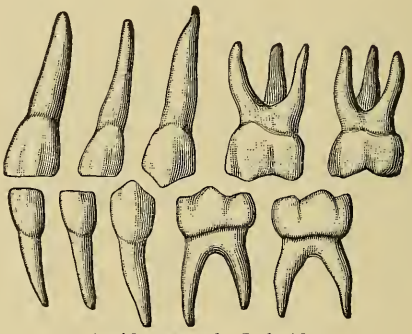


Calcification of the deciduous teeth. (Peirce.)

length, of dentin be formed than in the crown, but bone is to be formed beneath and about the sides of this root sufficient to hold firmly the, at present, loosely placed tooth.

Whatever the cause, as soon as root-formation begins the crown of the tooth commences its journey toward the gum-surface. It will be observed that the resorption of the bony impediment to the advance is upon the anterior edge (Fig. 66). When sufficient of the bony impediment has been removed, through the agency of osteoclasts, to permit the passage of the crown of the teeth, the gum-tissue is next resorbed, until the tip of the crown emerges and finally takes its anatomical position upon the summit of the jaw. The time at which this occurs differs with the classes of the teeth. As has been said, eruption is a phase of tooth-development beginning shortly after the completion of the crowns of the teeth, and it has been seen that the individual teeth differ as to the extent of their formation at a given time (Figs. 136). The teeth

Fig. 137.



Deciduous teeth. Left side.

make their appearance in the dental arches when root-formation is more than half complete.

Periods of Eruption.

As a general rule, the eruption of the deciduous teeth may be said to begin about the seventh month after birth, and is completed somewhere about the twenty-fifth month. This rule, however, varies within wide limits; some children may be born with teeth erupted; there are classical examples of this precocity—Cæsar and Richard III.;[1] again, the initiation of the process may not occur until the twelfth month or even later.

The incisor teeth are usually erupted in pairs, the molars and cuspids making their appearance in fours, the first molars in one group, the cuspids in another, and the second molars in a third group. The several groups require different lengths of time to complete their erup-

[1] *York.* Marry, they say my uncle grew so fast that he could gnaw a crust at two hours old; 'Twas full two years ere I could get a tooth. Grandam, this would have been a biting jest. *Richard III., Act II., Sc. iv.*

tion, the time occupied in the eruption of the first molars being longer than that required for the eruption of the other groups. Between the appearance of additional groups of the teeth an interval elapses, no doubt a physiological provision, for, as will be shown later, the process of dentition is usually accompanied by evidences of more or less local disturbance, frequently by disturbances throughout the intestinal tract, and even reflex disorders of the central nervous system occur. It is believed, therefore, that the period which elapses between the eruption of the dental groups permits the organism to recover from the effects of previous disturbance before the new source of irritation appears.

Table.[1]

Group 1.	Lower central incisors.	Time of eruption, 7 months.	Duration of eruption, 1 to 10 days.	Interval, 2 to 3 mos.
Group 2.	Upper central and lateral incisors.	Time of eruption, 9 months.	Duration of eruption, 4 to 6 weeks.	Interval, 2 months.
Group 3.	Lower lateral incisors.	Time of eruption, 12 months.		
Group 4.	First molars.	Time of eruption, 14 months.	Duration of eruption, 1 to 2 months.	Interval, 4 to 5 mos.
Group 5.	Cuspidati.	Time of eruption, 18 months.	Duration of eruption, 2 to 3 months.	Interval, 3 to 5 mos.
Group 6.	Second molars.	Time of eruption, 26 months.	Duration of eruption, 3 to 5 months.	

In the accompanying table it will be noted that the time of eruption of the lower lateral incisors is later than that of the eruption of the upper lateral incisors. The reverse course is frequently observed; indeed, it has usually been accepted as the rule of precedence in the United States. All tables as to periods of eruption give but the approximate times; while variations are extremely common, the ages given in this table are those at about which the several teeth may be expected to make their appearance. Stellwagen (the American editor of *Coleman*), in commenting upon this table, states that the periods of eruption in this country are from one-seventh or more earlier than the dates given. He suggests that the difference in food-habit may account for the differences in time.

Pari passu with the development and eruption of the teeth are developmental changes in all of the glandular appendages of the alimentary canal in such wise that alterations in their structure, and no doubt in their physiological chemistry, are accompanied by dental provision for the mechanical subdivision of foods of post-infantile character.

Symptoms of Dentition.

The process of dentition, like that of parturition, although unquestionably a physiological one, is even under normal conditions

[1] Coleman's *Dental Surgery* (Stellwagen).

attended by phenomena which would usually be called pathological. The general characteristic of all physiological processes is that they are carried on without that form of consciousness of the individual termed pain, a course rarely noted in either parturition or dentition. Within physiological bounds, therefore, we expect evidences of local disturbance as an essential part of the process of dentition. These are of a character which would be expected from a survey of the existing conditions. The erupting teeth are exercising pressure upon the tissues which overlie them and a process of resorption of these tissues is in progress. The parts about the tip of the erupting teeth are in a state of irritation. Normally the reaction to this irritation should not exceed a mild active hyperæmia, and probably there is a causal relationship between the hyperæmia and the presence of the cells, causing resorption of tissue. The vascular reaction is evident in the usual deepening of color over the site of the advancing tooth, and when the tip of the latter is almost visible, being overlaid by the epithelium alone, the gum beyond the tip is hyperæmic. The temperature of the part is elevated. As in all hyperæmic conditions, in its early stages relief is afforded from the irritation by pressure. It is common to find the infant instinctively seeking relief by pressing the fingers against the gum, and its mental irritation is appeased by having the finger of the attendant rubbed upon the gum over the advancing tooth. In the same manner biting upon hard substances, such as teething-rings, affords relief. Still more marked is the soothing effect of biting upon cold substances, such as ice which, in addition to mechanically lessening the local blood-supply, causes contraction of the dilated vessels. The parts about the erupting tooth give every evidence of being in a hypersensitive condition, not alone as a direct consequence of the hyperæmia, but also from direct pressure upon nerve-filaments. The evidences of nervous disturbance are both direct and indirect: the direct being those of local hyperæsthesia; the indirect are those indicating reflex disturbances, the most constant of which is usually a hypersecretion of saliva, the local disturbance reflexly stimulating the salivary glands to increased functional activity. Disturbances of the alimentary canal, while common, belong to pathological rather than to physiological dentition, as do also disturbances of the central nervous system.

Prognosis and Treatment.

Unless the process of dentition be accompanied by symptoms of greater severity than those above described, it will go through its course unaided, and calls for no interference. With the eruption of the molars and cuspids the irritation will be exaggerated, as these

teeth are usually erupted in series of fours, and present through their forms mechanical obstructions to eruption. While normally progressing dentition calls for no interference, it does emphatically demand that precautions be taken to prevent an abnormal course. The infant must be properly fed and nourished. If it do not appear to thrive upon the food it is receiving, the food should be changed, and if necessary again changed until it is found to subserve its purpose—sufficient nourishment. The question and problem of infant-feeding are fully discussed in works upon diseases of children,[1] and concern the general rather than the dental practitioner.

It is of the utmost importance that the alimentary canal should be kept in a physiological and aseptic condition. Even in cases where dentition appears to be pursuing a regular course the period is marked by various evidences of irritation and disordered function of the alimentary tract. Although there is not in all cases an evident causal association of teething with these disturbances, it is noted clinically that the symptoms disappear if treatment directed to oral causes be pursued. Assuming, as is warranted by evidence, that the oral condition attendant upon dentition will permit the growth in the mouth of micro-organisms which do not flourish there normally, and that their passage into the stomach and to the intestines is followed by various fermentations—decompositions—the products of which act as local and in some cases general irritants, intoxicants, or poisons, it is evident that the removal of the morbid conditions of the mouth, preventing by this and other means the development of organisms in the mouth, will materially lessen the abdominal symptoms. It is found clinically that antiseptic treatment of the oral cavity is followed by a lessening of the evidences of intestinal fermentation, which, of course, receive appropriate treatment also. These subjects, however, belong to pathological dentition; but presenting as minor symptoms during the course of apparently normal dentition, they demand and must receive attention.

A normal course of dentition may frequently be assured by proper precautions, which, if not taken, will be followed by a train of disorders, painful and uncomfortable, if not dangerous.

Pathological Dentition.

When the common symptoms noted become exaggerated the progress of the dentition may be deemed pathological.

SYMPTOMS.

The symptoms attendant upon pathological dentition cover a wide range, beginning as evidences of pronounced local hyperæmia, with all

[1] Starr, *Infant-feeding;* J. Lewis Smith, *Diseases of Children.*

of its attendant symptoms and consequences; the other extreme is attended by profound disturbance of the central nervous system, convulsions, coma, and death. That a fatal issue should follow or be consequent upon pathological dentition has been seriously questioned by most authors of works upon diseases of children, but cases have been observed wherein no other sufficient source of irritation was discoverable, and, furthermore, there are many cases on record where appropriate treatment directed to the dental organs has been followed by the disappearance of serious disturbances of the central nervous system.

According to the symptoms observable, which may furnish the only indication of the pathological conditions present, morbid dentition may be classed under two heads: first, cases in which all disease-conditions appear to be local; secondly, those in which other and distant organs give evidence of disorder. These may again be subdivided according to the severity of the local and general symptoms.

Local Symptoms.—Instead of the normal localized hyperæmia of the gum, with its hypersensitivity and reflex increase of secretion, evidences of active inflammation may be observed: the gum assumes a dusky hue; if the apex of the tooth be about to penetrate the gum, the epithelium may be swollen and the area of vascular injection be larger. The tissues are swollen, the temperature is elevated, and the child gives evidence of painful local disturbance, notably by fretful crying, and by seizing the breast or bottle-nipple and quickly releasing it as soon as bitten upon, indicating pain upon pressure. The sleep is broken, and if the symptoms continue the child soon suffers from insufficient nourishment. The readiness with which the child will take cold substances, ice or ice water, is notable and self-explainable. Pressure and rubbing of the gum at a point distant from the focus of irritation afford relief. These symptoms usually subside with the appearance of the tooth through the gum, although in case of erupting cuspids local disturbances frequently persist until the entire crown is erupted.

In the more marked cases of local disturbance evidences of bacterial infection of the mucous membrane of the mouth make their appearance, such as ulcerative stomatitis; while, as a rule, the breaking down and ulceration of the tissue are confined to the parts overlying the erupting teeth, a general stomatitis or widely scattered patches of ulceration may make their appearance. The localized condition has been called odontitis infantum.

General Symptoms.—Intestinal Disturbances.—It is with cases of this type and with those of greater severity that disorders of the alimentary canal are commonly associated. These may express themselves in diarrhœa, with discharges of a "chopped spinach" character, in a serous

diarrhœa, but in some cases obstinate constipation may be noted. It is the rule, even in the milder of these cases, to find the stomach reject food immediately or soon after feeding. In the diarrhœal cases excoriation of the anus is frequently found, indicating the irritating nature of the alvine discharges. In addition, the child suffers from the pains of colic—*i. e.*, spasmodic contraction of the intestinal muscles and the generation of gases. All of these symptoms clearly point to abnormal fermentative changes occurring in the contents of the intestinal canal, and as clearly indicate the therapeutic measures likely to be useful.

Skin Disorders.—It is so common as to be almost termed the rule to find at the time of these intestinal symptoms that there are eruptions observable on the skin. The mildest form of these is a herpetic eruption about the mouth; in other cases papular and vesicular eruptions are observed upon the skin of the body and limbs.

Pulmonary Symptoms.—The close association of the fifth and tenth cranial nerves is frequently cited in dentistry, notably in connection with shock from operations; another expression of it may be noted in the laryngeal cough which sometimes attends the eruption of groups of teeth, and which disappears upon their eruption.

Nervous Disorders.—Disorders referable to the central nervous system are the most alarming, and are those indicating the higher grades of severity of irritation. The milder forms of these disturbances are faint muscular twitchings. The child wakes out of sound slumber with an irritable cry, and is soothed with the utmost difficulty; it dozes, only to be awakened again. In these cases there are usually other morbid symptoms, such as the disturbances of the alimentary canal described above and the evidences of oral affection. These symptoms may occur without any such evident connection with the teething-process that a probable causative association with dentition is not made out. In fact, in the absence of evident local irritation the association of teething with the general disturbances is frequently denied, but in refutation of such a position it is noted that treatment based upon morbid dentition as the cause of the nervous disturbance is commonly followed by a disappearance of the latter. There is much doubt as to the precise nature of the dental irritation in such cases, as there is no morbid condition of the parts overlying the erupting teeth which would serve to explain the irritation. It is assumed with good reason, therefore, that the irritation is of the pulp itself; that the tissue of the pulp is being subjected to pressure through the processes of tooth-building and tissue-resorption not proceeding harmoniously. The source of irritation has been by some authors ascribed to an inflammation of the follicle. Certainly such an inflammation might occur in the tissues immediately surrounding the tooth; but in that event we should expect

to find associated disorder of the overlying tissues, which is frequently not the case.

A distressing symptom not easy to elicit on account of the age of the patient is headache. The child is sleepless, and cries without apparent cause; it becomes quiet, partially from exhaustion, and after a period again commences sobbing. The indication of central disturbance may at times be noted in the contracted pupils of the eye and in throbbing arteries. The usual treatment, the administration of chloral hydrate and potassium bromid, with cold applications to the head, furnishes relief, which is frequently not complete without attention to the dental organs.

In the more severe and dangerous cases the evidences of disorder of the central nervous system become unmistakable. These appear as clonic convulsions or symptomatic eclampsia. While it is probable in many cases that reflex irritation from the process of dentition in itself is but a secondary cause of convulsions, yet evidence is sufficient to warrant its being regarded as a determining factor. In very many cases teething-convulsions appear to indicate a neurotic family taint, and eclampsia may attend many disorders in children of this type, notably the mechanical and chemical irritation induced by the presence of large masses of indigestible food in the intestines.

So-called teething-convulsions occur usually at a time when several —an abnormal number—teeth are in process of simultaneous eruption. The onset of the convulsions is rarely, although apparently often, sudden. If the child be closely observed, it is noted that a period of cerebral disturbance—fretful crying, evidences of headache, sleeplessness, etc.—is followed by a period of dulness and somnolence, or the child may lie with eyes half-open. Twitching of one or more groups of muscles may be observed; the orbicularis oris and other muscles of the lips, and the muscles of the eye, notably the superior and internal recti, may contract spasmodically. A common muscular spasm ushering in convulsions is that of the adductor muscles of the thumbs; the thumbs are drawn toward the palms of the hands. The adductor muscles of the feet contracting, the feet are drawn inward. This period may be ushered in by a sharp cry, the eyes roll upward with the lids half-open, and consciousness is lost. The symptoms may disappear, the child awakening dazed and fretful; or it may sink into sleep. Unless the source of irritation be removed, or active therapeutic measures be instituted, the eclampsia may return and in severe cases be the precursor of death.

TREATMENT OF PATHOLOGICAL DENTITION.

There is no means for assuring a normal and smooth progress of dentition comparable in value with hygienic precautions. If intelli-

gent care be taken of the clothing and diet of the child, together with measures to insure normal digestion, and due regard be paid to ventilation, in the vast majority of cases the period of dentition will be passed through with but trivial oral disturbance. In all cases of pathological dentition, therefore, a careful superintendence of these matters must form an essential feature of the treatment of the case.

Treatment of Local Disorders.—Since conditions of hyperæmia invite the development of micro-organisms, it is evident that sterilization of the mouth must play an important part in the treatment of the irritation incident to teething. The importance of this measure is further emphasized by the researches of Miller,[1] who found that numerous forms of organisms associated with intestinal fermentations are present in the mouth, and develop when conditions favorable to their growth appear; passing into the alimentary canal, those not destroyed by the acid gastric juice pass into the intestines and give rise to fermentations of the intestinal contents.

The feeding-nipple, which should be of the simplest type, should be well washed in hot water after each use, then washed in a dilute antiseptic solution, and be kept submerged in it until needed. Each feeding-bottle should be sterilized in boiling water after using. The practice of the nurse rubbing irritated gums with the finger should be discountenanced, as should also the use of teething-rings which are permitted to lie about and take up infective material. If hard-rubber, ivory, or metallic rings be used, they should be carefully sterilized by boiling.

To reduce local hyperæmia of the gum above an erupting tooth a common domestic measure is valuable, viz., a small block of ice is placed in a clean napkin, and confined in place by a knot; the infant places it in its mouth at pleasure if old enough, or the nurse permits the child to bite upon it. The mechanical effect of biting upon a hard substance has added to it a degree of cold which lessens the local vascular engorgement.

It should be noted that during the period of dentition children who are vaccinated against smallpox have the suffering incidental to teething much increased.

More marked degrees of vascular engorgement, hence irritation and suffering, usually demand the relief by local bloodletting and lessening mechanical resistance following the operation of gum-lancing (see later).

Should general stomatitis, with or without stomatitis ulcerosa, make its appearance, the mouth is to be promptly and freely sprayed with a 3 per cent. solution of pyrozone, followed by a spray of potassic chlorate, gr. xx–℥j, which usually affords prompt relief. Should the spots of ulceration not disappear promptly, the mouth and tissues about the

[1] *Micro-organisms of the Human Mouth.*

ulcer are to be guarded by soft linen napkins; each ulcer is dried and touched with carbolic acid, full strength. The spraying is to be repeated at intervals of three hours during the waking period. This method of treatment is productive of decidedly better results than follow the use of the common formula of honey and borax.

When evidences of active inflammation exist in the gum overlying an erupting tooth the indication is free gum-lancing with a blade of such razor-like sharpness that no pressure is exercised in the operation. Conjoined with this measure, to which is added spraying with pyrozone, steps should be immediately taken to empty the intestinal canal of its contents, as not infrequently constipation is an attendant condition. The constipation incident to some cases of stomatitis or gum inflammation (gingivitis) may be induced or aggravated by the administration of soothing-syrups, which usually contain opium in some form. The same result is produced by the domestic use of soothing-syrups and paregoric (tr. opii camphorata) to relieve diarrhœa; by lessening the sensitivity of the bowel and decreasing its peristaltic movements irritating substances are retained in the bowel, a menace to the child's health. It is not intimated that opium in minute doses (and a one-drop dose of tr. opii deodorata is a very large one for an infant of eight or ten months) is not a valuable drug under some conditions, but its use should follow that of laxatives and intestinal antiseptics, not precede them.

Treatment of Skin Eruptions.—The eruptions which appear upon the skin during dentition may be a source of annoyance to the child by causing itching. As a rule, measures directed toward a regulation of the intestinal functions cause a disappearance of the skin affections. If the eruption be widespread and cause much itching, a wash of phénol sodique, diluted to one-third with water, usually affords relief. If the surfaces be then dried and talc powder dusted over them, the condition is much alleviated. About the mouth and over excoriated surfaces an ointment of zinc oxid is useful.

Treatment of Intestinal Symptoms.—These, in the vast majority of cases, call for two measures—evacuation and the use of antiseptics, the character of both evacuant and antiseptic to be determined by the nature of the case. It has been held that diarrhœa during dentition is due to a heightened irritability of the bowel, a reflex irritation, and sedatives have been administered to lessen such hypersensitivity. That such a heightened sensitivity may be present and that abnormal progress of dentition may produce an irritable condition throughout the intestinal tract, are quite probable, but experience has shown, and modern pathological theories indicate forcibly, that the bowel-condition is caused by the action of irritating products of fermentation upon the walls of the intestine. As emphasizing the importance of prompt and effective

therapeusis in this connection, it must be remembered that not only are the normal intestinal functions interfered with as a consequence of these fermentations, but a train of disturbances follows. First, normal digestion, both gastric and intestinal, is incomplete; instead of forming nutrient solutions, the food undergoes abnormal fermentations, the products of which act as local poisons disordering the function of the bowel. Although absorption is lessened, it does not cease, and poisonous substances are taken into the portal circulation. There is no doubt that many of these poisons are chemically destroyed in the liver, but some of them may find their way into the general circulation and cause symptoms of toxæmia. In any event, the formation and absorption of assimilable pabulum are greatly lessened, and debility—*i. e.*, lessened vital resistance—is induced. The products of abnormal fermentations act as local irritants, causing irritation of localized or widespread areas of the intestine. Concentrated at some one portion of the length of the canal, they may cause spasmodic contraction of its muscular wall, the painful condition of colic. Other fermentations, or the same perhaps acting over large surfaces, induce irritative diarrhœa. So long as these conditions are permitted to exist all bodily functions suffer, and if dentition be in progress its course is interfered with. The probable causative connection between mouth and intestinal fermentations has been discussed.

Recognizing the cause of the condition in the presence of irritating substances in the intestine, the rational indication is to free the bowel from them. There is perhaps no laxative comparable with castor oil for this purpose. Children take castor oil better than adults. After the action of the oil, additional fermentation is checked by the use of antiferments—antiseptics. Preparations of thymol, eucalyptus, benzoic and boric acids, such as listerine, are extremely useful in this connection; ten drops in water may be administered every three hours. Both powders and solutions given to children may be sweetened by the addition of a minute quantity of saccharin, which has the additional advantage of being an intestinal antiseptic. The subsequent administration of magnesium hydrate is useful; it has the advantage of allaying irritation by coating the mucous membrane; and if acids be present, they combine with the magnesium, forming faintly laxative salts. To lessen great irritability and procure quiet a few drops of paregoric may be added to the magnesium fluid. Compound chalk powder—mist. pulv. cretæ co.—is also used for this purpose.

Treatment of Nervous Conditions. — Should the premonitory symptoms of disturbance of the nervous system make their appearance, attention should at once be directed to a search for the cause. If constipation exists, the bowels should be freely opened; magnesium

citrate in effervescing solution is well taken. Clothing is loosened, cold applied to the head, and an examination of the gums made. If a tooth, or, as is more frequently the case, several teeth are in process of eruption at one time, their crowns partially or completely imprisoned by overlying gum, lancing should be done at once. More severe symptoms call for more active treatment, the general therapeutic principle involved being the lessening of the cerebral blood-supply. This is most quickly accomplished by placing the child almost waist-deep in water as hot as can be borne and pouring cool water over the head, when, as a rule, the symptoms promptly subside. After immersion, a rectal injection of glycerin (one drachm) will usually cause a free stool. To insure a period of cerebral quiet it is advisable in the more severe cases to administer a cerebral sedative, a bromid preferably, which, if combined with chloral hydrate, which children bear well, will induce sleep:

℞. Chloral. hydrat., gr. ij;
Sodii brom., gr. v;
Starch paste, ℥ij.—M. (Atkinson.)
S. To be administered per rectum.

After sleep, if appearances indicating dental irritation be observed, gum-lancing is practised.

It has been repeatedly noted that where evidence of marked cerebro-spinal irritation is present, for which no probable source can be assigned and an examination of the gums shows no apparent local disturbance, yet if it be at a period when one or more teeth are in process of eruption, but are still covered or bound down by gum-tissue, if gum-lancing be practised, relief is immediate and may even avert a threatened attack of eclampsia. It is presumed that these are cases of pulp-irritation in which a failure to sufficiently remove tissue in advance of the tooth-crown has caused pressure upon the pulp from below.

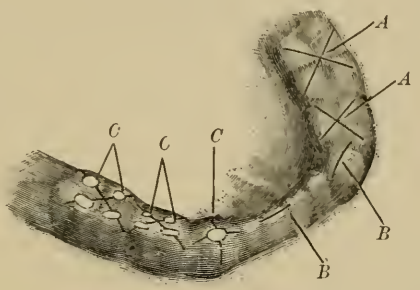
Gum-lancing.—The operation of gum-lancing consists in making incisions through the gum-tissue overlying developing teeth in such manner as to remove mechanical obstruction to their advancement. The direction and character of the incisions are governed by the form of the advancing tooth (Fig. 138).

The gum over the incisors and cuspids is divided on a line parallel with the cutting-edges of the teeth; the cut over the upper incisors should be a little outside of the teeth-edges; that over the lower incisors slightly inside, so as to induce normal positions, the upper teeth outside of the lower.

When the point of the cuspid has penetrated the gum-tissue the

crown is still confined, owing to the conical form of the tooth, so that to effectually free it radial cuts may be necessary (*C*, Fig. 138). The gum over the molars, with the exception of the upper first, is divided in X-form, the cuts extending from cusp to cusp. The upper

Fig. 138.



Lines of incision in lancing: *A*, *A*, over the molars; *B*, *B*, over the cuspids and incisors before eruption; *C*, *C*, over the molars and cuspids after partial eruption.

first molar having but faintly marked cusps, and only an outer and inner cutting-edge to the tooth, is best freed by a crucial incision.

The instrument to be used for dividing the gum is a pointed bistoury of extreme sharpness; its edge is to be wrapped in linen or cord until only a half inch from the point is exposed. This precaution is taken to avoid accidental wounding of the mouth. The child is to be placed and held in such position that the light shall fall upon the jaw to be operated upon, and so that no movements shall be permitted. It is advisable—almost necessary—to have an assistant hold the child in the proper position so that it cannot move head, body, or limbs. A position favorable for operating is to have the child lie upon its back between the knees of an assistant, the left hand of the assistant holding the hands of the infant, the right hand being used to steady the head. In another position the child sits upon one thigh of the assistant, the back of the head resting upon the chest, and the hand of that side (usually the right) pressed upon the child's forehead to hold the head firmly. The other hand and forearm hold the child's arms and hands firmly.

The operator encloses the gum about the part to be cut with the thumb and forefinger of the left hand, so that the bistoury cannot slip and cut lip, cheek, or tongue. Incision over the erupting tooth should be made until the knife-blade is felt to touch the enamel-surface. The operation of scarifying the gums, making merely a few scratches to relieve engorged vessels, is but temporizing with the condition; the cut should be of sufficient extent to entirely remove tension from above the tooth. The little finger of the right hand may rest upon the chin of the child as an additional guard.

In case the crown of an erupting molar is confined by a block of tense gum attached upon two or more sides, the entire block is to be removed; the block is drawn tense by means of traction with a tenaculum piercing the flap, and three sides freed by means of the bistoury point; the final cut may be made with scissors of extreme sharpness.

While it is true that in many cases children struggle under this operation, and give evidence that it is a painful procedure, gum-lancing is frequently followed by immediate tranquillity upon the part of the child; fretfulness disappears and nursing is resumed immediately after the operation, which occupies but a few seconds of time.

More or less bleeding follows upon the operation, which, as a rule, ceases spontaneously. A short period of bleeding is desirable, so that vascular engorgement may be reduced. Suckling by the infant usually serves to check the bleeding; the tissues about the cut surfaces are compressed by tongue and lips during suckling, and bleeding ceases. In the event of the bleeding continuing the mouth should be carefully examined, and a piece of ice in a napkin may be given the child to suck. Obstinate bleeding may require the use of styptics, but these should be of a character to cause only coagulation of the blood, not the destruction of tissue. A little powdered tannin laid upon the cut acts promptly, as does also a small amount of powdered alum.

CONSTITUTIONAL STATES MODIFYING DENTITION.

Children who are the victims of hereditary syphilis usually cut their teeth very early, the alveolar process being in many cases insufficient. Cases are recorded where children have been born with crowns of teeth visible upon the gum, there being no evidence of root-formation, the crowns being loosely held to the gum by fibrous tissue. It is necessary to remove these loose crowns to permit the infant to suckle. Children affected with rachitis have the process of eruption much delayed. It is seen, therefore, that the presence of loose crowns of teeth is a condition pointing to, though by no means diagnostic of, hereditary syphilis. Also, that long-delayed eruption of teeth should prompt a search for further indications of rachitis. Particularly in children in whom a history of hereditary syphilis is obtainable the process of dentition may be accompanied by rapid and frequently widespread breaking down of the soft tissues over and about erupting teeth. Local measures of treatment seem to be of but little avail, except that antiseptic treatment undoubtedly prevents complications from extraneous infection.

In children classified indefinitely as strumous, which may mean the children of syphilitic or tuberculous parents, or those with no such history whose surroundings are of the most unhygienic kind, the process of dentition may not only have an untoward course, but phagedenic

ulcerations may occur. It is usually in the degree of a child's debility, either inherited or acquired through improper care, that dentition assumes morbid features. The treatment of such cases must be directed to raising the health standard. As local therapeusis, no measures seem more effective than the sprays of hydrogen dioxid first; next, potassium chlorate, and, if conditions indicate it, sprays of dilute listerine, which is stimulant, antiseptic, and slightly astringent.

Infantile Scurvy.—Cases are recorded[1] in which the improper feeding of children has been followed by evidences of scorbutus. The gums become tumid, and hemorrhagic extravasations occur in their substance; the periosteum is stripped from the margins of the alveolar walls, the soft tissues hanging in discolored pendulous masses about and beyond the teeth if any be erupted. Local treatment proving ineffective, the scorbutic nature of at least one of these cases was made clear by the disappearance of the oral disorder when the patient was placed upon an antiscorbutic diet.

Second Dentition.

The period of second dentition begins without disturbance of the temporary denture, by the eruption of the first permanent molars, posterior to the second temporary molars. At the period when this occurs, from five to seven years, all of the permanent teeth are in process of formation, even the third permanent molars, although none other of the permanent teeth except the first molars will normally appear in the dental arch for a year, or in some cases two years. The order in which the permanent teeth make their appearance varies, but the following table shows the approximate dates at which they may be expected to appear in the dental arch:

First Molars,	5½– 7	years.
Central Incisors,	7 – 8	"
Lateral Incisors,	8 – 9	"
First Bicuspids,	10 –11	"
Second Bicuspids,	11 –12	"
Cuspids,	12 –14	"
Second Molars,	12 –15	"
Third Molars,	16 –20	" and indefinitely beyond.

With the exception of the permanent molars, all of the teeth have deciduous predecessors, the bicuspids being the successors of the temporary molars.

At the time of eruption of the first permanent molars calcification

[1] E. C. Kirk, Proc. Odontological Society, New York, *Dental Cosmos*, 1895.

has been for some time complete in the roots and alveolar process about the roots of the temporary teeth, and the roots of the deciduous incisors are already beginning to be absorbed, *pari passu* with the development of the corresponding permanent teeth. The permanent teeth are all at different periods of formation, differing in extent according to groups of fours: (1) central incisors, (2) lateral incisors, (3) first and (4) second bicuspids, (5) cuspids, (6) second molars, (7) third molars—group (1) being the first molars (Fig. 139). The figure exhibits the stage of tooth-formation—*i. e.*, the extent of calcification at successive periods. As calcification proceeds in the permanent teeth having deciduous predecessors the latter suffer, first, loss of apical alveolar walls, and next gradual loss of root-substance, in exact correspondence with the growth of the permanent teeth. The lines in the illustration (Fig. 140) show the extent of root-loss of each tooth at successive periods.

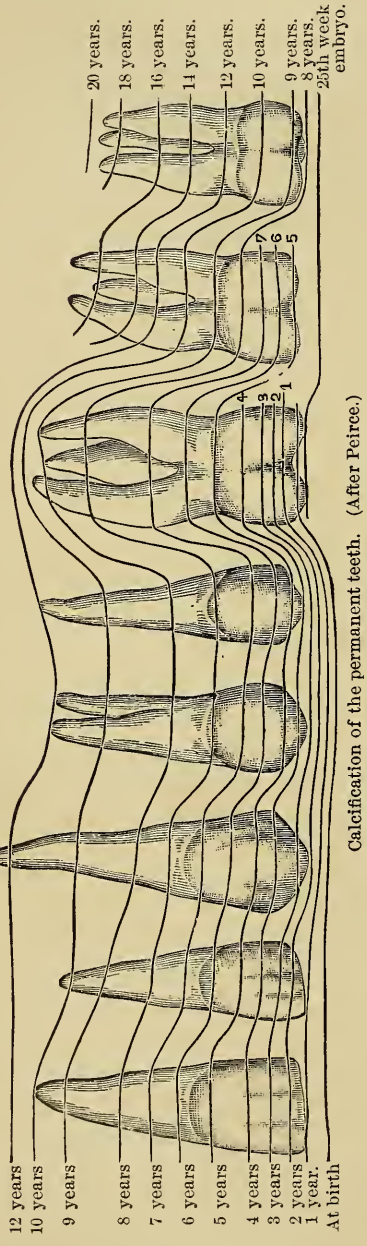
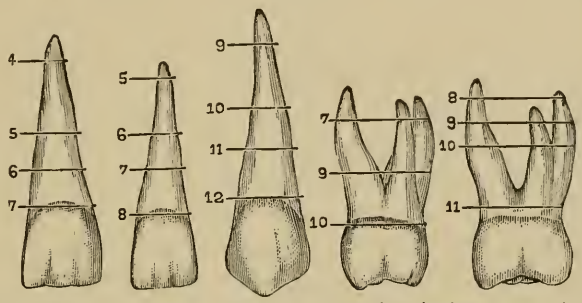


Fig. 139. Calcification of the permanent teeth. (After Peirce.)

To comprehend the nature of the processes involved it is essential to obtain an accurate view of the anatomical conditions existing prior to and during the loss of the deciduous teeth and the appearance of those of the permanent denture. The teeth, both temporary and the partially formed crowns of the permanent teeth, lie in that portion of the bone called the alveolar; that is, the portion of the lower jaw above the inferior dental canal, and in the upper jaw below the infra-orbital foramen. The cuspid crowns in both jaws are more deeply seated than are any of the other teeth, and in the lower jaw are deeper than the mental foramen. Before

resorption of the roots of the deciduous teeth begins the temporary teeth occupy a segment of alveolar bone of nearly as great a radius as that required for the permanent teeth, but the depth of which, from infra-orbital or mental foramen to the alveolar margins, is decidedly less than that of the adult jaw. In the base of this segment, separated from the bodies of the bones by bony lamina and from the pericemen-

FIG. 140.



Decalcification of the deciduous teeth. The numbers indicate years. (Peirce.)

tum of the temporary teeth by a layer of bone, lie the developing permanent teeth, with the exception of the molars, which lie in the posterior portion of the alveolar bone, which at the period under consideration (about five years) is of slight extent compared with its adult size, both as regards depth and length. Its length, from the distal wall of the temporary second molar to the base of the coronoid process, is but little more than sufficient to easily accommodate the crown of the permanent first molar; its depth between gum-surface and the roof of the inferior dental canal is but little more than sufficient to accommodate the height of the crown.

The buccal cusps of the crowns of the developing bicuspids lie in the triangular pyramidal depressions between the roots of the temporary molars, separated from the roots, as are all of the other crowns from the roots of their deciduous predecessors, by laminæ of bone and pericementum. The crowns of cuspids and incisors lie inward lingually of the roots of the deciduous teeth.

ERUPTION OF PERMANENT FIRST MOLAR.

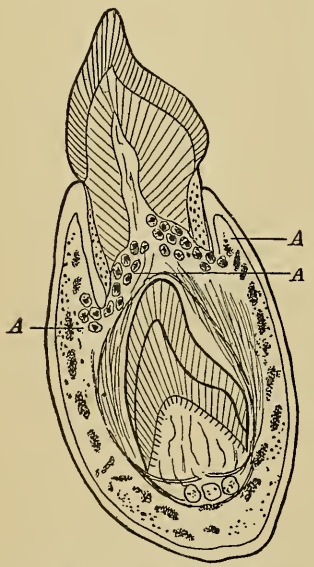
This tooth, vulgarly called, from the time of its eruption, the sixth-year molar, has a process of eruption similar to that of the deciduous teeth. Being the largest tooth of the dental series, the passage of its large crown requires the resorption of an extensive amount of gum and alveolar tissue; this process is usually accomplished in the course of a few months. It differs in no respect from the physiological eruption of the temporary tooth. It is to be recalled that in the development of the jaws two divisions of the bone are recognized, the alveolar

portion and body portion; normally their development proceeds together and harmoniously. With the growth of the alveolar process which forms about the roots of the permanent first molars the distances from the alveolar border to the levels of infra-orbital and mental foramina increase—*i. e.*, the alveolar bone is growing in height throughout its length. This growth normally causes a separation of the anterior teeth, which is to be regarded as the precursor to resorption of the roots of these teeth.

RESORPTION.

In advance of the crown of the tooth in process of eruption a number of large multinucleated cells make their appearance, whose office it is to effect the removal of tissue interfering with eruption. The origin of these cells is not known; by some they are believed to be transformed osteoblasts, by others, leucocytes. The latter is the more probable explanation; that is, these cells are phagocytes, named from their particular function osteoclasts, or bone-destroyers; those attacking and removing the roots of teeth are called odontoclasts. In the light of present physiological theories it is believed that these cells have a secretion capable of digesting bony tissue and dentin; that is, they remove molecularly the tissues interfering with eruption. It is possible that they secrete a ferment or solvent which effects the solution of bone and dentin, the solution being removed by the lymphatic system. As the result of the presence and life of these cells, first, disappearance of the bony lamina between the crowns of the permanent and the roots of the deciduous teeth occurs; next, the roots of the latter are attacked when the permanent crown impinges upon the deciduous root. It is possible that this element of pressure, of advance of the permanent crown, may be the cause of the presence of these multinucleated cells (see Chapter I., Reproduction of Cells). These cells lie between crown and root, and as the latter is removed the former advances. It will be observed from the relationship of parts that resorption does not begin at the very apex of the root. Those portions of the alveolar process which immediately surround the roots of the temporary teeth also undergo resorption, a new process being built about the roots of the permanent teeth (Fig. 141).

FIG. 141.



Showing the relations of an erupting permanent tooth to its deciduous predecessor: *A*, *A*, *A*, odontoclasts.

The process of eruption of the permanent second molars is the same as that of the first molars. The development of the jaws increasing the distance between the distal wall of the first molar and the base of the coronoid process, proceeds until there is space enough between the second molar and the coronoid process to admit the crown of the third molar, which erupts late, by a process similar to that of the other permanent molars. Not until this eruption is complete do the maxillæ acquire their full adult forms.

DISORDERS OF THE SECOND DENTITION.

Provided that nothing occurs during the period of second dentition to interfere with the normal resorption of the roots of the temporary teeth, the process of dentition as regards the twenty-eight anterior teeth proceeds in an orderly manner. Given such interference, abnormalities of the second dentition make their appearance; many of these will be discussed in the succeeding chapter, under the head of malpositions. It will be recalled that the teeth are an evolution of the dermoid system, which fact possesses pathological significance in certain acute and specific skin diseases. It is noted in some cases of the eruptive fevers of children, particularly when the child is much debilitated, that after the cessation of the acute disease a necrotic affection of the jaw occurs, involving the alveolar bone and its contents. As many of these cases occur between the ages of three and seven years, the temporary teeth are still *in situ;* these with the partially developed permanent teeth and the enclosing bone may be exfoliated. The necrotic process may involve but one tooth, or may include all of the temporary teeth, their successors and a large mass of bone.[1] The disease with which this necrosis is most frequently associated is scarlet fever;[2] it is also found as a sequel of measles and smallpox. It will be observed that all of these diseases are forms of specific dermatitis, and the teeth as part of the dermoid system are affected (see Chapter XI.). "The cases prior to exfoliation of the bone exhibit a stripping of the periosteum, apparently beginning about the necks of the teeth. A discharge of pus having a fetid odor is present, and the soft tissues may be raised from the bone for a variable extent;" that is, there is evidence of purulent periostitis. In the course of some weeks, six or eight, the necrotic bone and its contents exfoliate. Salter observes that the sequestra forming after severe scarlet fever are much more extensive than those which form as a sequel of measles.

Treatment.—In the absence of any guide as to the amount of bone which has died, the operator is compelled, as in all cases of necrosis, to adopt palliative measures, the most important one being the free and

[1] Salter, *Dental Pathology.* [2] *Ibid.*

frequent use of antiseptic washes and sprays—hydrogen dioxid. The prescription of Garretson for such cases should be applied to hasten exfoliation and to stimulate still vital parts—*i. e.*, enough tincture of capsicum and myrrh (tr. capsici et myrrhæ) is to be added to a glass of water to cloud the water; this is then to be used freely as a stimulant and astringent mouth-wash. As soon as the sequestrum is loose it should be removed; the parts heal by granulation, provided due attention be paid to the general physical welfare of the child.

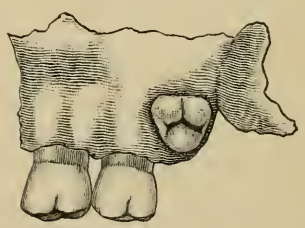
Non-resorption of Temporary Roots.—The failure of resorption of the roots of temporary teeth will necessarily interfere with the eruption of the permanent teeth. Perhaps the most common cause of failure of root-resorption of the temporary teeth is to be found in septic conditions of their roots. Whether the pulp of a tooth performs any active office in the process of root-resorption is not known; the evidence that it does is but imperfect and indirect, so that for the reasons of failure of resorption search must be made outside of the pulp-chamber. The pulp of a temporary tooth dead as the result of invasion of caries undergoes decomposition; infection of the pericementum occurs, and septic pericementitis results. An abnormal condition is established, and resorption does not occur; the conditions are not favorable for the presence and activity of odontoclasts; should they be present, their vital activities are poisoned by the products of the micro-organisms present—instead of functionating they die. An irregular resorption does, however, occur over some portions of the roots. If the purulent condition be removed and the parts be made aseptic, resorption occurs but is delayed. It is evident that the underlying permanent tooth is imprisoned until, by a loss of overlying structures, it is free to move into position. Many of these dead teeth and dead roots are removed by a process similar to that which occurs in the casting off of the useless roots of the permanent teeth; a general resorption of the provisional alveolar bone occurs, and the root is cast out; its separation from the soft tissues may again be accomplished by the process of suppuration.

Treatment.—If a pulpless deciduous tooth be present in the dental arch when the corresponding permanent tooth of the opposite side of the jaw is in process of eruption, the pulpless tooth should be extracted. Prior to this time the deciduous tooth should be retained, provided, by appropriate antiseptic treatment, its pericementum can be brought to and be maintained in a healthy condition. The space between adjoining teeth occupied by the temporary teeth affords room for the accommodation of the permanent successors. Too early extraction is followed by insufficient space. This is particularly notable following premature loss of the deciduous second molars.

Eruption of Molars.—It is rare that abnormalties are associated with the progress of the eruption of the first or second permanent molars. It may be noted in very rare cases that there is a failure of harmony in the development of alveolar and body bone; that insufficient space exists posteriorly for the accommodation of the second molars. They suffer more or less impaction, particularly in the lower jaw; as a rule, subsequent development of the body of the bone affords the needed space. It is with the eruption of the third molars that aberrations are most frequently noted.

Pathological Eruption of the Third Molars.—At the time of eruption of the permanent second molars, the third molars in both jaws occupy recesses in the alveolar bone and are in but a partial state of development. In the upper jaw these recesses occupy the tuberosity of bone bounded anteriorly by the distal walls of the second molars, and posteriorly by the tips of the pterygoid processes of the sphenoid bone. This rounded prominence grows larger as the development of the teeth proceeds. It is unusual that much lack of space exists for the accommodation of the tooth. Should the development of the tuberosity not keep pace with that of the tooth, and eruption proceed, the direction of eruption is outward toward the cheek; the relations of the pterygoid process and of the palate bone with the tuberosity make a path of greatly increased resistance at the palato-alveolar angle; backward eruption is prevented by the pterygoid processes; direct eruption, by the second molar and pterygoid process, so that the path of least resistance is outward toward the cheek. It is occasionally noted that this tooth erupts with its occlusal face pointed directly toward the cheek, a position at right angles to that it occupies normally (Fig. 142). In rare instances the upper third molar is caught between the second molar and the pterygoid process; in such cases eruption is prevented or delayed (see Chapter X.).

Fig. 142.



Abnormal eruption of the upper third molar.

In cases of difficult eruption conditions may arise which tend to increase the morbid processes and to alter their character. There is, first, an irritative hyperæmia of the soft tissues surrounding the erupting tooth. As this is the region of the mouth least affected by dental cleansing agents used by the patient, and in which the irrigating office of the saliva is very incomplete, fermentation-processes are common; their products added to a part in active hyperæmia can but aggravate the condition. Pyogenic cocci, usual inhabitants of the mouth, may infect the tissues, finding with numerous other organisms a habitat

between the tooth and the swollen gum-margin; thus it is that pus-formation may accompany the delayed eruption. If the swollen parts be incised, and antiseptic sprays be directed into all of the recesses, a disappearance of symptoms is noted.

It is in connection with the lower third molar more frequently than with any of the permanent teeth that pathological eruption is most common. In civilized races the room for the accommodation of the crown of the lower third molar, between the distal wall of the second molar and the base of the inner root of the condyloid process, is frequently but scant; the tendency of maxillary development is to render restricted space the rule rather than an exceptional occurrence. The crown of the tooth does not make its appearance until developmental changes have secured sufficient space for it. This may delay its eruption for months or years, and in some cases prevent eruption altogether. In others the development of the tooth may proceed after all developmental changes in the jaw have ceased, and the teeth are entirely encapsuled (see Chapter X., Impacted Teeth). The delay in eruption in the more severe cases is due to improper configuration of the bony surroundings; the delay in milder cases may be due to imprisonment by soft tissues; in other cases pathological conditions are caused not so much by imprisonment as by irritation of the soft tissues.

It is almost the rule that eruption of the lower third molars is attended by some degree of discomfort. Usually for some months before the cusps of the crown make their appearance through the gum there is an ill-defined uneasiness about the jaws and some stiffness of the muscles of mastication. These symptoms disappear and recur at irregular intervals until the crown is entirely through the gum. Interference is rarely called for; the usual antiseptic mouth-washes employed by the patient guard against infection of the irritated parts. In cases where the gum presents a swollen and irritated appearance relief follows the application to the affected gum of iodin tincture. Should the mesial half of the crown be free and the posterior half covered by a curtain of gum, it is advisable that the pocket between the tooth and gum be eradicated, as it forms a recess in which the active causes of dental caries thrive, and in which fermentations arise, forming irritating products. In the more severe cases these may induce active inflammation of the soft tissues to an alarming extent. In all of these cases where bands or curtains of gum-tissue are the cause retarding eruption the indication is to freely divide the tissue longitudinally. The precaution should be taken to spray the parts freely with an antiseptic before and after making the incision. The cutting may be rendered painless by slipping under the gum-flap for a few moments a pledget of cotton containing a 6 per cent. solution of cocain hydrochlorid, the

same solution being painted over the flap. The incision is made with a pointed bistoury, its point being directed toward the crown.

It is when some portion of the posterior segment of the crown is held back by bony tissue that the more severe symptoms appear, usually in the degree of bony imprisonment. The general character of the pains in these cases is heavy and rheumatic, indefinitely located about the angle of the jaw and the ear. Combined with pain is usually marked stiffness of the masseter muscle; in the more severe cases the spasmodic contraction of the muscle may simulate trismus of that side. These symptoms may appear irrespective of any marked evidences of inflammatory reaction of the tissues around the imprisoned crown. The pains are of the type called periosteal—dull, gnawing, heavy.

The pockets between gum and tooth-crown offer passageways for infection of deeper tissues, so that it is not surprising, particularly in unclean mouths, that inflammation and suppuration of the tissues about the part may occur. The inflammatory process may extend to the neighboring soft tissues; there is usually increased rigidity of the masseter muscle, so that it is with the utmost difficulty that the mouth can be opened even with mechanical assistance. It is not unusual, therefore, as a result of inability to reach the parts for local therapeutics, for suppuration to extend far beyond the original focus of irritation. Relief is not attainable until free vent for the pus is given, the discharge being often through the cheek.

TREATMENT.—Nowhere more than here does the dictum of surgery —remove the source of irritation, apply. The source of irritation common to all of the cases being the tooth for which there is insufficient room, the general indication is its extraction. This operation is, however, frequently attended with difficulties which render it in some cases highly impracticable. In the least severe cases of bony imprisonment, if untoward symptoms can be held in abeyance for some months, the obstruction to the tooth's advance may be removed and eruption be completed. The principal object during the period of delay is to prevent infection of the parts, so that antiseptic mouth-washes play an important part in the treatment. Local irritation may be largely allayed by the free application of tincture of iodin diluted with alcohol.

If the rheumatic pains about the jaw and neck and the stiffness of the masseter muscle be of moderate degree, relief is afforded by free massage of the parts. If the pains assume a neuralgic character, much relief may be secured through the use of sedative ointments, such as those of aconitia and veratria.

℞. Aconitia, gr. j;
Cerat. simp., ʒj.—M. (Flagg.)

In making this ointment it is to be manipulated so that it is certain the drug is evenly distributed throughout the cerate. A minute portion of the ointment is rubbed over the masseter muscle in front of the ear and about the angle of the jaw by means of the finger.

Flagg commends the efficacy of an ointment of veratria when that of aconitia fails to procure relief:

℞. Veratria, gr. xx;
Cerat. simp., ʒj.—M. (Flagg.)
S. Used in the same manner.

It should be noted that both of these ointments are actively poisonous, and should be kept from absorbent surfaces.

Should severe symptoms arise, extraction is imperatively demanded, and had best be done before increasing stiffness of the muscles interferes with the proper opening of the mouth. In some cases there is no doubt of the practicability of extracting the third molar; when the crown is freely exposed and not decidedly imprisoned by the coronoid process, forceps may grasp it firmly and by an inward and upward rotary movement the tooth is extracted. In cases of more decided impaction, the resistance offered outwardly by the external root of the coronoid process, backward by the internal root of the same, and forward by the second molar, may render extraction impossible without the previous removal of some portion of the bone. It is evident that in such a case attempts at forcible extraction would be inevitably followed by fracture of bone or of the tooth, the latter occurring more frequently than the former. It is advisable—indeed, almost necessary—in such cases that the second molar be the tooth extracted. This applies also to the cases of more decided imprisonment with active inflammation.

Where stiffness of the masseter muscle fixes the jaws so that they cannot be separated sufficiently to secure a grasp upon either the third or even the second molar, mechanical violence may be necessary to separate them. A jaw-separator is placed in position, the patient etherized, and the separator operated until sufficient space is gained to secure a grasp upon the tooth; failing the third molar, grasping the second molar. The muscle is sometimes of board-like hardness, and resists the force of the separator. Indeed, such resistance may be offered that separation can only be effected by fracture of the muscle or of the bone. In these cases an antiseptic spray—hydrogen dioxid—is directed against and about the third molar for a period of ten minutes or longer; as soon as sterilization is assured a curved bistoury is used to divide the

swollen gum-tissue about the tooth, and cold compresses are placed over the angle of the jaw. The antiseptic spray is subsequently applied at intervals of about two hours. If local massage be practised in addition, the swelling and muscular hardness usually disappear in a few days, when the jaws may be separated sufficiently to allow extraction of the molar (see Impaction of Teeth, Chapter X.).

CHAPTER X.

MALFORMATIONS AND MALPOSITIONS OF THE TEETH.

In the majority of persons the entire denture consists of thirty-two teeth arranged in two parabolic curves, one upper and one lower, each semi-denture having sixteen teeth. Normally both the upper and the lower teeth are bilaterally symmetrical, the eight teeth of either half of the upper or lower denture corresponding exactly in size, form, and position with the corresponding eight teeth of the opposite side. Each tooth of each upper and lower eight, while differing in size and details of form from all of its fellows, yet bears a definite relationship to each of the others as regards size.

Abnormalities of the teeth are found associated with position, size, form, and structure. Aberrations in form, structure, and size are included under the head of malformations of the teeth; aberrations of position are discussed under the head of malpositions of the teeth. The particular section of dentistry relating to malpositions of the teeth is by general consent made a special department of operative dentistry, that of orthodontia; but many of the phases of the subject are of great pathological interest, although the therapeutic measures usually demanded are mechanical in character and clearly belong to the fields of operative and prosthetic dentistry.

Malformations of the Teeth.

Malformations of the teeth may be either macroscopic or microscopic; they may be evident to the unaided eye, or may require for their detection special preparation for observation under the microscope.

MICROSCOPIC MALFORMATIONS.

Microscopic or histological defects of the teeth may affect any of the dental tissues, enamel, dentin, cementum, pulp, or pericementum.

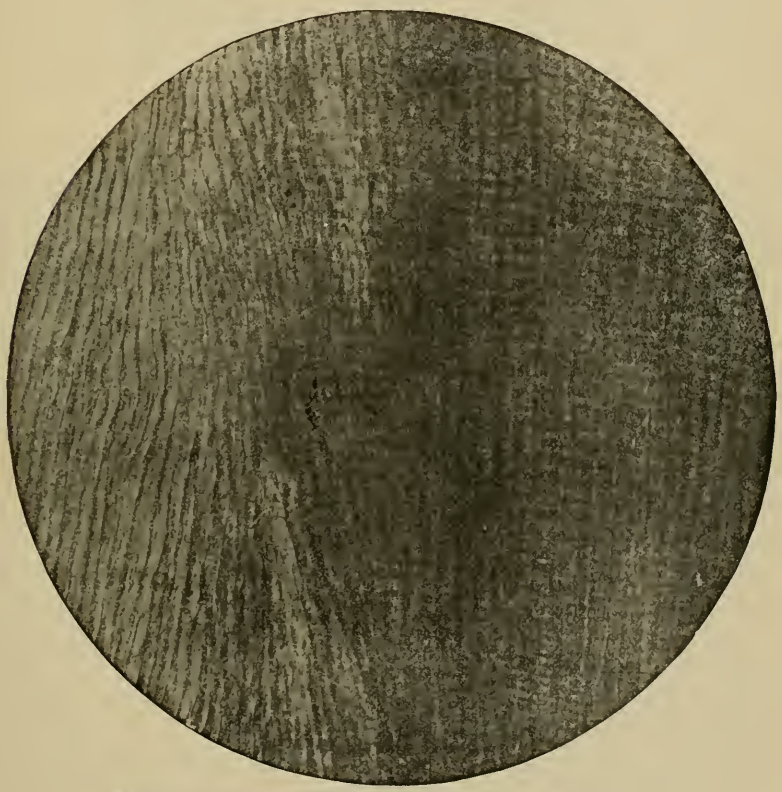
Enamel.—Defects in enamel-structure range from any degree of orderliness in the even distribution of globular bodies and cementing-substance in the tissue to gross aberrations in formation. The finer variations of structure are not easily recognizable.

Theoretically perfect enamel should show in longitudinal section a series of squares of uniform size built into rods, the spaces between the squares and rods being marked by lines of cementing-substance having

a refractive index slightly different from that of the squares. While such a structure is perhaps never found, it is difficult to draw a line where aberrations from such a standard become pathological. An arbitrary standard might be assumed as follows: regard any enamel as pathological where areas of it differ from its general substance to such an extent as to have a decidedly different refractive index. A typical form of abnormality is noted in what are known as white spots of the enamel, areas in which an opaque surface exists instead of the normally translucent enamel.

In Fig. 143 is represented a magnification of 500 diameters of a section made of a portion of a white spot where it adjoins normal

FIG. 143.



Margin of section through "white spot." × 500. (Williams.)

enamel. The area of white spot when viewed by transmitted light shows dark in the section. The enamel of the white spot is more or less granular in structure, and its globules are not fused together in rods as in normal enamel. Williams suggests as an explanation of this condition that there is a deficiency of the cementing-substance.

Fig. 144.

Antero-posterior section of human lower molar, showing defects of structure in the vicinity of sulci. × 12. (Williams.)

Fig. 145.

Section of human molar, showing dentinal fibrillæ penetrating enamel. × 600. (Williams.)

Enamel-formation about the sulci of teeth is frequently faulty; owing to an imperfect union of the enamel-segments forming the cusps of the teeth minute fissures exist in the enamel; these are most marked in the fissures of molars, as shown in Fig. 144. The enamel bounding these fissures has an irregular structure.

The dentinal fibrillæ may penetrate the substance of the enamel (Fig. 145), occupying defined channels in its substance; this is a developmental accident. Williams points out that the organic filaments from the dentin become atrophied with the progress of enamel-formation and canals remain. The probable explanation of this condition is that a process of an odontoblast prior to enamel-deposition finds its way through the soft pre-enamel-deposit, and enamel-deposit encloses it, calcification occurs, and a permanent defect results. This condition, as also many other variations of structure found in the dental tissues of man, are shown by Williams to have their normal prototypes in the

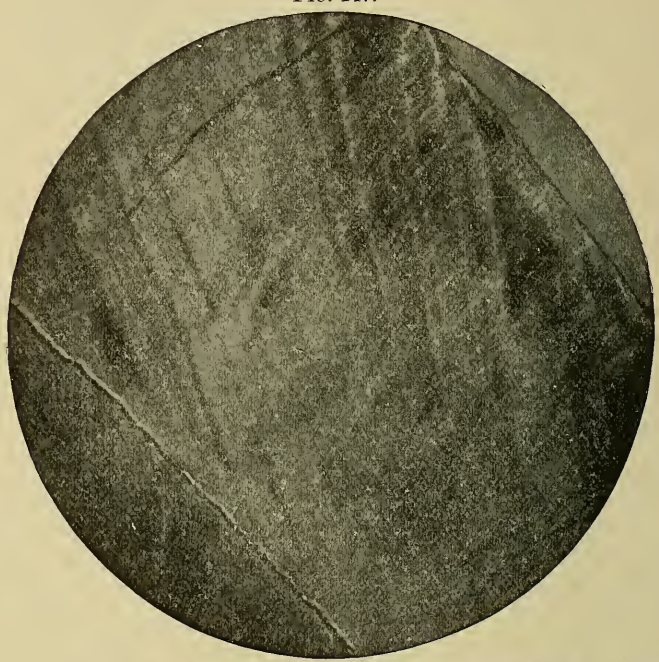
FIG. 146.

Section of human bicuspid, showing mass of very imperfectly calcified enamel projected into the dentin, with coarse fissures leading to the surface. × 75. (Williams.)

dental tissues of lower animals; for example, the penetration of dentinal fibrillæ into enamel is a normal condition in the teeth of the kangaroo. The presence of organic tissue in the enamel of man is, however, always to be regarded as pathological. Such conditions are not to be confounded with fissures of enamel where large lines of faulty calcification or non-calcification extend through the thickness of enamel (Fig. 146).

Enamel, even normal enamel, is not of uniform composition ; were it so, it would exhibit, in addition to an orderly arrangement of its histological elements, a uniformity in color. So common are differences in this direction that the presence of pigment-bands must be regarded as normal. It is the rule to find enamel traversed by deeply pigmented parallel bands (Fig. 147) which pass obliquely upward from the surface

FIG. 147.



Section of human incisor, showing "bands of Retzius" and marked stratification of enamel. × 125. (Williams.)

of the dentin to the surface of the enamel. These are termed the bands of Retzius (see Chapter VII.); they appear to mark the size of the enamel-cap at successive periods of its growth.

Stratification and striation of the enamel, as shown by Williams, must be regarded as normal physiological records of the mode of enamel-formation.

All of these histological defects represent variations of deposition, of formation, no doubt due to fluctuations of the nutritive processes of the child at the time of tooth-formation. Histological records made in the enamel are not like those made in other tissues, for there is no nutritive provision through which such defects can be remedied at subsequent periods.

Profound nutritive disturbances, such as those attending hereditary syphilis in children, affect the structures of the teeth. One of the gross

results of this disease is a common malformation of the general form of the incisors. The hard tissues of such teeth exhibit microscopic evidences of faulty histology ; they are dull and opaque and traversed by irregular bands. Viewed in section the enamel of such teeth is seen to be almost structureless (Fig. 148). Williams found that the contents

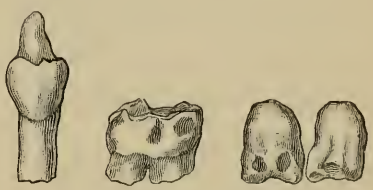
Fig. 148.

Section of enamel from syphilitic tooth, with appearances resembling the lacunæ of cementum. ×600. (Williams.)

of the large irregular spaces in this enamel did not respond to stains—*i. e.*, did not contain organic matter.

There is evidence that other forms of specific dermatitis—scarlet fever and measles—which occur at an early age may affect the formation of enamel. The defects attributed to the exanthemata are irregular pits upon the crowns of, particularly, the incisors (Fig. 149). With a history of a case, including the age of the child at the period of the disease, if examination be made of the positions of the defects, the age will serve as an indication as to whether there has been any connection between the eruptive fever and the dental malformation. For example, if enamel-pits upon incisors have been caused by an eruptive fever between the ages of four and five, they should occupy about the half-way area of the crown-face ; it is evident that the enamel being already formed about the cutting-edge

Fig. 149.



Teeth of eruptive fevers.

of the tooth, alterations of nutrition could not affect the already formed tissue. In cases where a causal association of enamel-defects with the eruptive fever is made out with reasonable clearness it is usual to find all of the crowns of the teeth which are in process of formation affected in a similar manner. The defects and pittings are so pronounced in some of these cases as to give a general honeycombed appearance to the crowns; such teeth are known in dental parlance as "honeycombed teeth."

Cases are observed where there has been a formative crisis to the extent of having no enamel whatever formed over the occlusal section of the crown, its deposit on the remainder of the crown being quite normal (Fig. 150).

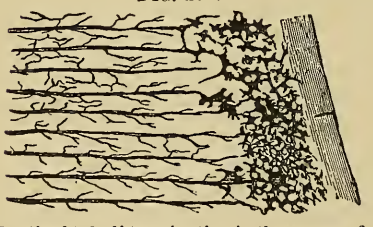
Fig. 150.

D. B. Freeman[1] records the case of an individual, aged twenty-six years, whose teeth anterior to the second molar were entirely devoid of enamel. The condition was hereditary, it appeared in both brothers and sisters, and could be traced back for three generations.

Dentin.—Data regarding the finer phases of defective histological structure of the dentin are meagre. It has been observed that the dentinal tubuli of some teeth are much larger than in others of the same age, and, no doubt, future investigations with an improved technique directed toward a study of the exact mode of dentin-formation will exhibit defects more certainly.

The chief histological defects noted in dentin are areas of faulty or non-calcification, called interglobular spaces (see Chapter VII.). These are most common in the dentin immediately underlying its covering tissue, so common in the dentin under the cementum that this portion of dentin has been called the stratum granulosum, the granular layer of Tomes (Fig. 151). In the body of the dentin these spaces have a more irregular distribution.

Fig. 151.



Dentinal tubuli terminating in the spaces of the granular layer. (Tomes.)

In wet ground-sections (Röse) the dentinal filaments are seen to pursue an unbroken course through these areas. The contents of the interglobular spaces react to stains like the sheaths of Neumann; that is, they probably contain transitional tissue. These areas probably represent, as do defective spots of enamel, periods of depressed vitality or of altered nutrition. In the light of present knowledge regarding the subject they are to be viewed as areas in which the calcific process was faulty. The malformations noted in

[1] See Guilford, *American System of Dentistry*, vol. iii.

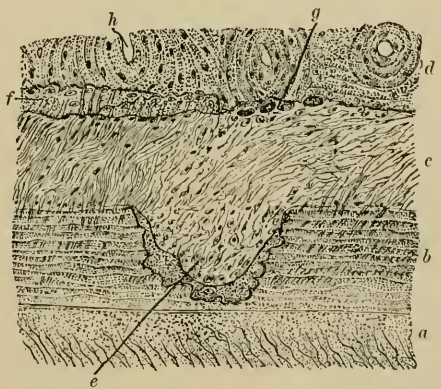
connection with the enamel of syphilitic teeth have their analogues in the dentin (Fig. 152).

Histological congenital malformations of the pulp have not been

Fig. 152.

Section showing interglobular spaces in dentin of a syphilitic human tooth. (Williams.)

Fig. 153.



Section of a bicuspid with its alveolus, showing a pit-like absorption upon the side of the root in which the redeposit of the cementum has begun: *a*, dentin; *b*, cementum; *c*, peridental membrane; *d*, bone forming the wall of the alveolus; *e*, absorbed area of cementum. It will be noticed that a new deposit of cementum has begun the filling of the area, and that the soft tissue in the area of absorption is of a cellular type. The bone also shows the effects of absorption in the cutting away of portions of the rings of the Haversian systems at *f*, while at *g* the presence of osteoclasts shows that absorption is in progress at that point. (Black.)

recorded, the normal histology of the organ not being made out with sufficient certainty to determine what appearances are to be regarded

as abnormal. Grosser aberrations, such as those shown in Fig. 103, are made out.

Cementum.—As stated in Chapter VII., the pericementum contains numbers of multinucleated cells—odontoclasts; and their presence is not to be regarded as abnormal. The cementum of the roots of teeth may exhibit evidences of former action of these cells in excavations of cementum which by a subsequent deposition of cementum have become filled. This gives an irregular course to the cement-laminæ (Fig. 153). These appearances are to be regarded as not necessarily pathological, for the following reason: for some time (years) subsequent to the eruption of the teeth developmental changes occur in the alveolar bones, depositions (subperiosteal) increasing their volume, are accompanied by resorption of other portions of the bone, such a balance being kept between the processes that the teeth, although shifting their positions, are kept in normal occlusion.

MACROSCOPIC MALFORMATIONS.

The teeth may vary from normal either as regards size or external configuration.

Variations as to Size.—It is patent to the most casual observer that the teeth vary as to size. Comparisons in this direction are made by an examination of the upper central incisors. Fig. 154 shows nearly the extremes of observable sizes; Guilford[1] points out that excessively large central incisor-crowns are usually supported by abnormally small conical roots. Marked giantism of the central incisors usually occurs in pairs, the other teeth being of normal size. On the other hand, dental giantism of less degree may involve all of the teeth of a denture. The molar teeth are occasionally of enormous size, the bicuspids rarely so, and the cuspids next in frequency to the molars as to the occurrence

Fig. 154.

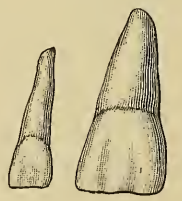
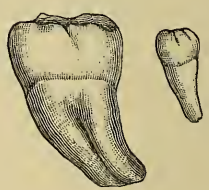


Fig. 155.



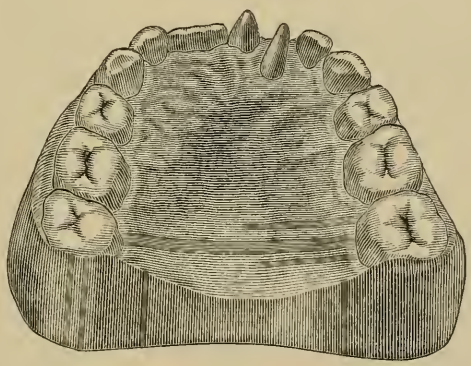
of giantism. Guilford observes that giantism of the cuspid-crowns, unlike that of the central incisors, is usually accompanied by a corresponding size of root. He mentions the case of a cuspid measuring an inch and a half in length from tip to tip.

[1] *American System of Dentistry*, vol. iii.

Dwarf Teeth.—Deficiency in size is of more common occurrence than excessive size. It appears to occur more frequently with the upper third molars and upper lateral incisors than with any other teeth. The accompanying figure (155) shows the extremes in size between two perfectly formed lower third molars. The stunting of these and of other teeth is, however, usually associated with such an aberration of outward form that most dwarf teeth must be considered as abnormal in form as well as in size.

Abnormalities of Form.—It is in the outward forms of teeth that the greatest aberrations are met with. These range from a slight exaggeration of one or more of the architectural elements of a tooth to such malformations as produce an entire unlikeness to all tooth-forms—to masses which cannot be properly classified as teeth. The most common of these malformations is an irregularity of cusp-form; cusps are suppressed or are but primitive. A survey of the primal basis of tooth-forms reveals that most of these malformations are due to improper modifications or combinations of the formative cones. A common type is one or more of the incisors, most frequently the lateral incisors (Fig. 156), presenting as unmodified cones or as conical masses, the normal form, it will be recalled, being a truncated and compressed cone.

Fig. 156.

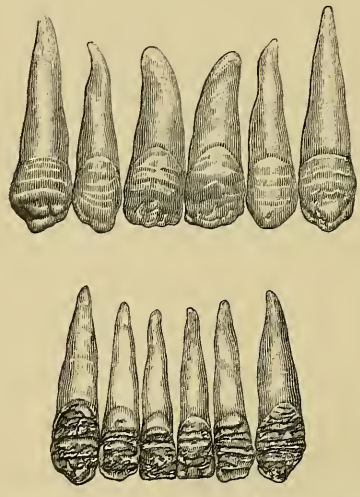


Next in point of frequency, the upper third molar is seen to consist of but a single cone instead of three fused cones. At the other extreme the primitive cones may be so combined as to produce such irregularities of cusp and sulcus arrangement that the masses have lost all semblance to normal tooth-forms.

Pitted and Grooved Teeth.—The effects of profound disturbance of the course of nutrition upon the histological structures of the teeth have been described. Such teeth are also affected as to their outward form. The malformations may consist of series of irregular grooves and pittings, the crowns having the general normal outlines;

or, again, the form of the tooth itself may be altered (Fig. 157). A single groove across the face of a tooth represents, no doubt, a period of nutritive disturbance, but cases are seen where there is a repetition of grooves separated by ridges of apparently normal enamel, indicating waves of nutritional disturbance, as shown in Fig. 157. The defective formation may be confined to one portion of the crowns of the teeth, most commonly the occlusal half of the first permanent molars and of the incisors.

Fig. 157.

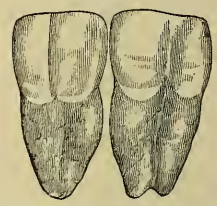


Showing the front teeth grooved from the alternation of perfectly and imperfectly developed portions of enamel. (Tomes.)

Fig. 158.

Fused molars.

Fig. 159.



Permanent central and lateral incisors of the upper jaw, united throughout the whole length of the teeth. (Tomes.)

Fused Teeth.—It is occasionally observed, more frequently with the upper second and third molars than with other teeth, that, instead of two separate teeth terminating the dental series, its extremity is occupied by a large dental mass, which upon examination shows the cusp-elements of both the second and third molars present. Upon extraction this mass is seen to form but one tooth, but an anatomical analysis clearly reveals its dual character; its anterior portion corresponds with a second molar, its posterior with a third molar. The root-arrangement also indicates a fusion of two teeth (Fig. 158). The anterior teeth may be united after the same manner (Fig. 159). It will be observed that this union is most likely to occur where the adjacent tooth-follicles have least anatomical separation from their fellows; it is most common between incisors and incisors, incisors and cuspids, and second and third molars. It appears not to have been observed in connection with the bicuspids, the crowns of these teeth during the formative stage being

confined between the roots of the temporary molars. The follicles of teeth liable to fuse depend for separation from one another upon the formation of bony walls.

A histological examination of these teeth exhibits a pulp-chamber, which may be common throughout, or, as more frequently happens, distinct and separate root-portions of the pulp are observed. The malformations of the crowns are clearly traceable to the fusion of the lateral walls of the enamel-organs before or soon after enamel-formation begins, because it is found that these teeth have a common pulp-chamber. If the fusion occurred subsequent to the first deposition of calcic substances, the pulp-chamber would be double. In the period of root-formation the common pericementum may or may not be divided; if it is, the fused crowns are associated with separate roots; if not, the roots are fused and have dentin common to both throughout.

Concrescence of Teeth.—Concrescence of teeth is their union after the tooth is formed; it is evident, therefore, that the union can only be caused by fusion of cementum. This means that at some portion of the formative and eruptive period the bony partition between the teeth disappears, and that their pericementi become united, receding from the line of compression as cementum is deposited between and joining the roots.

In the eruption of the third molars, particularly the upper, temporary lack of space for the eruption of the crown may cause absorption of the bone covering the roots of the second molar, and fusion of the formative pericementum of the third molar with that of the second occurs; a deposition of cementum then binds the teeth together, preventing the eruption of the third molar.

Geminous Teeth.—These have been termed twin teeth, in contradistinction to fused teeth. In twin teeth the enamel-organ, developed

Fig. 160.

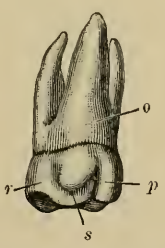


Fig. 161.

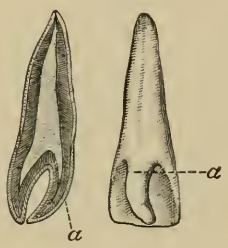


Fig. 160.—The upper molar.

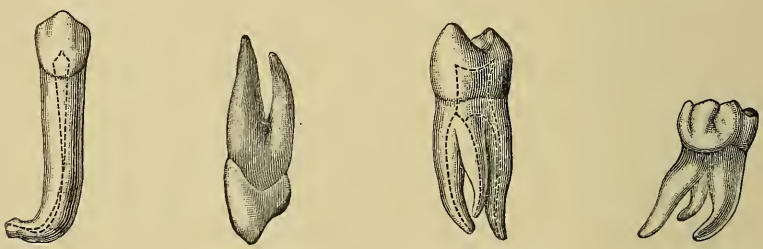
Fig. 161.—Showing unusual development of the cingule or basal talon on an incisor. (From case reported by W. H. Mitchell, *Dental Cosmos*, vol. xxxiv., p. 2036.)

from some *single cord*, assumes the form of two teeth, and the tooth developed in such enclosure has the form of a duplicated prototype. The second segment is to be classified as a supernumerary mass.

Supplemental Cusps.—It is occasionally observed that a tooth has a greater number of cusps than normal. The most common form of this condition is a supplemental mass attached to the palatal side of the mesio-palatine cone of the upper first molars (Fig. 160). The palatal tubercle, the prominence upon the cingule of an upper incisor, may be of exaggerated size. In one case recorded (Fig. 161) this development gave the appearance of a talon upon the tooth, a distinct cusp-segment in itself.

Malformations of Roots.—Differences in regard to the size, arrangement, forms, and number of the roots of teeth are the most common of dental malformations. The roots of teeth may be abnormally short; they may be inordinately long—roots of cuspid teeth more than an inch in length are by no means uncommon (Fig. 162). The roots of cuspid teeth may be bifurcated (Fig. 163), and those of bicuspids trifurcated (Fig. 164). The lower molars may have three roots (Fig. 165). Thompson[1] has pointed out that such malformations, together

Fig. 162. Fig. 163. Fig. 164. Fig. 165.



Upper cuspids. Upper first bicuspid. Lower first molar.

with certain crown malformations, are instances of zoölogical atavism. Abnormalities of root-forms are of extreme frequency. Examples of these are seen in Figs. 166–177.

Fig. 166. Fig. 167. Fig. 168. Fig. 169. Fig. 170.

Upper molars (Ottolengui). Lower bicuspids.

Hutchinson Teeth.—Attention was first called to, and an adequate explanation of the condition given by Jonathan Hutchinson, as to the effect of hereditary syphilis upon the permanent teeth. He observed in the children of syphilitic parents a malformation of the anterior

[1] *American Text-book of Operative Dentistry.*

teeth, the incisors commonly and inconstantly the cuspids. The situation of these malformations is such as to correspond to that period of

Fig. 171. Fig. 172. Fig. 173.

Abnormalities in teeth.

Fig. 174. Fig. 175. Fig. 176. Fig. 177.

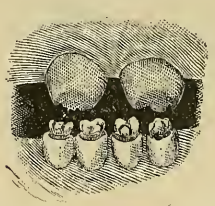
development when the evidences of hereditary syphilis are noted in the infant.

Confusion of description by dentists and faulty observation by medical practitioners as to both forms, have led to much confusion as to what particular forms of teeth are to be regarded as syphilitic. The teeth most frequently affected are the upper central incisors. It will be recalled, in this connection, that children of syphilitic parents have usually a tardy eruption of the deciduous teeth. The teeth have a dull, opaque color. The central or lateral incisors upper and lower, either, both, or any of them, have, instead of the normal angles and flattened curves of the labial faces, a roughly rounded and stunted appearance; the occlusal edge of the tooth is narrower than its neck. Over the tips of these stunted and conical teeth the enamel is irregularly and badly formed; but there is a semblance of the three enamel-tubercles found normally. The middle tubercle appears to be of the most defective enamel (Fig. 178), because it is soon lost by abrasion, leaving a notch in the tooth at its former site (Fig. 179). While Hutchinson regarded the central incisors as the diagnostic teeth of hereditary syphilis, all of the teeth undergoing amelification at the same time may exhibit deformities, one of the most frequent being the malformation of the cuspids.

It has been noted that not all children who are the victims of hereditary syphilis present these dental appearances; and, again, ap-

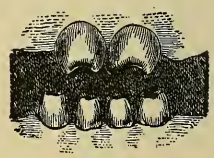
pearances *said* to be identical with them are observed in children *said* not to be syphilitic; nevertheless the presence of such teeth is usually regarded as a valuable diagnostic sign of hereditary syphilis. Thera-

Fig. 178.



Syphilitic teeth in upper and lower jaws as they appear when recently erupted.

Fig. 179.



The teeth of hereditary syphilis at maturity.

peutic measures based upon this, as well as other doubtful indications, are followed by better results, perhaps, than when the general indication of the dental malformation is ignored.

The existence of interstitial keratitis is accepted as additional diagnostic sign of hereditary syphilis in the infant.

Odontomes.—In rare cases dental masses of such irregular form as almost to defy classification make their appearance in the dental arch. They may appear instead of the teeth, in addition to them, or after them; such masses may be grouped under the head of odontomes. In some instances they never make their appearance in the dental arch, but may remain imbedded in the substance of the jaw for lengthened periods; here they may give rise to cyst-formations (called odontoceles), may excite no evident reaction, or may be the exciting cause of various morbid reactions.

In some instances the fusion of two teeth may produce a mass of such irregularity of form as to give the appearance of a dental tumor, but critical examination rarely fails to demonstrate a fusion. Equally odd appearances may result from the fusion of supernumerary with the normal teeth (Fig. 180). The nature of these cases may usually be made out by the more or less orderly arrangement of cementum and enamel (Fig. 181). A specimen (Fig. 182) in the museum of the Academy of Stomatology of Philadelphia, shows two masses making their advent between the upper central incisor teeth, forcing these teeth aside. From the appearance of the surfaces of these masses they represent the results of a plication of the surface of the enamel-organ from

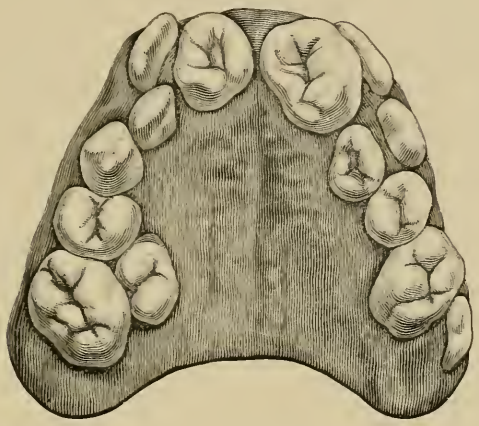
Fig. 180.

Fig. 181.

A supernumerary tooth attached to the roots of an upper molar, the supernumerary tooth being inverted. (Smale and Colyer.)

which they derived their enamel. Clearly they are the results of the formation of two adventitious dental cords. Fig. 183 illustrates an odon-

Fig. 182.

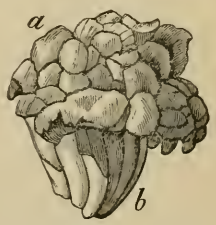


toma in which the dental nature of the growth is to be clearly made out; the enamel-forming organ from which the mass derived its enamel-cup was of anomalous form. Such specimens are known as warty teeth (Salter).

Broca[1] was the first to offer a systematic classification of tooth-tumors, although the connection between various tumor types and dental tissues had long before been made out.

The discussion of the pathogenesis, clinical history, and treatment of odontomes which arise from some portion of the tooth-follicle in its embryonic state and cause the formation of extensive neoplastic growths which bear no resemblance to tooth-forms, their only points of association being scattered histological appearances, belongs to the province of general surgical pathology, and their treatment to general or special surgery, so that they can be fitly dismissed from these pages with a brief mention.

Fig. 183.



Odontoma. (Garretson.)

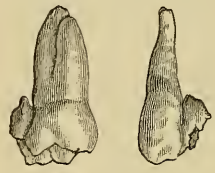
A developed tooth-follicle contains within it both epiblastic (epithelial) and mesoblastic (connective) tissue-elements. An aberrant, morbid tissue-development may, therefore, give rise to either epithelial or connective-tissue new formations, the cellular elements of which may be of an embryonic (sarcomatous) or mature (fibromatous, osteomatous, etc.) type; or may be the starting-point of either comparatively benign epithelial growths or even of carcinoma. The growth may contain the elements of several types

[1] *Recherches sur une nouveau groupe de tumeurs désigné sous le nom d'odontomes*, 1867.

combined. It appears that cyst-formation most commonly results from a continued collection of fluid between the epithelial coating of a tooth-follicle and the dentinal and enamel elements underlying, the accumulation of fluid causing the formation of a sac lined by the transformed epithelial wall.

Odontomata of direct clinical interest to the dental practitioner are those connected with some portion of a tooth, showing an irregular or anomalous growth of some one or more of the dental tissues.

Fig. 184. Fig. 185.



Enamel excrescences. (Salter.)

Fig. 186.

Results of hernia of a pulp. (Salter.)

Many of these may be and have been classified under the head of malformations of the teeth. Many of the surgical odontomata exhibit but slight trace of any dental structure, but in the class under discussion it is

Fig. 187.

Fig. 186 magnified.

evident that a continuous relationship has been kept between an enamel-organ, a dentinal pulp, and a cementoblastic structure, even though the aberrations of tooth-form and of limitations as to extent of growth

diverge widely from the normal. In some of these growths it is evident that tooth-development has progressed in an orderly manner to a varying degree before any aberration of development occurred; in others it is evident that development has been aberrant from the beginning.

The growth may be associated with enamel, dentin, or cementum development; and its tissue-elements as regards the dentin may remain normal, or may occasionally partially revert to other types, vaso-dentin, osteodentin, etc. Figs. 184 and 185 exhibit the results of activity of the enamel-organ continued after its normal formative period and in an irregular manner.

The development of both enamel and dentin may proceed in an orderly manner for some time, when an irregular developmental impulse arises in the dentinal pulp, leading to its enlargement and extension beyond the enamel-organ, which latter structure suffers atrophy, and a growth of the following type results. The pulp outgrowth may not occur until both crown and root of the tooth have been formed in an orderly manner, when a hypertrophic impulse causes this organ to extend far beyond its normal boundaries, still, however, enclosed in the follicular wall; the pulp in its new relations deposits dentin, over which cementum is deposited (Figs. 188, 189, and 190).

Fig. 189.

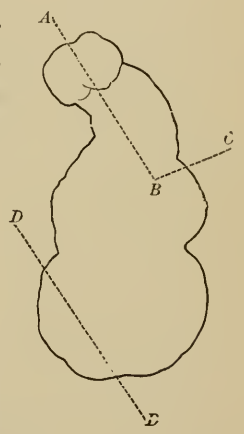


Fig. 188.

Figs. 188 and 189.—Results of pulp-hernia. (Tomes.)

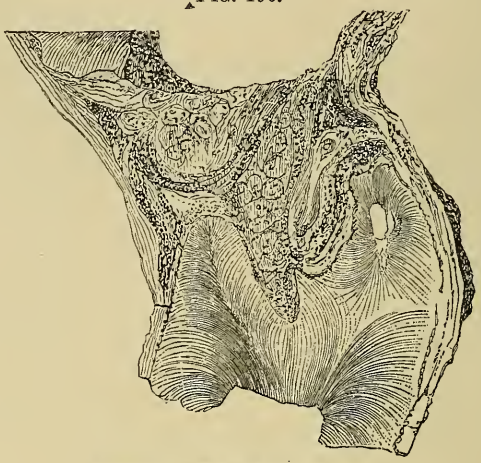
Neither the enamel-organ nor dentin-pulp may assume its normal type, and yet the relationship between the epithelial tissue of the enamel-organ and a layer of odontoblastic cells in the underlying mesoblastic tissue is maintained, together with a general enclosure in a follicular wall, in which event an irregular mass (see Fig. 183, warty tooth) containing the dental elements is formed.

Irregularity of growth is undoubtedly more frequently associated with the cementum than with any other dental tissue. It may assume the form of a generally excessive deposit, be in the form of a nodule or nodules, or be a large irregular mass.

Treatment.—The treatment of these cases is that applied to all

removable tumors—radical extirpation. While in some cases this consists of the operation of tooth-extraction on a large scale, in others the

FIG. 190.



A section of Fig. 189 through *A B*.

removal of an odontome involves the performance of a surgical operation of some magnitude.

Anomalies of Number.—Although the dental series of man normally consists of thirty-two members, cases are frequently observed where the number is in excess of or less than that number.

DEFICIENCY.—It is observed with some frequency that the upper lateral incisors of a denture never make their appearance, a condition traceable to the influence of heredity in some of the instances. The permanent cuspids erupt and occupy the lateral incisor space. The third molars may never appear; instead of being represented by a rudimentary tooth, they are apparently never formed. There is no doubt that in some of the cases of apparent absence of the third molars that the teeth may be encysted in the maxillæ; but when none of them appear up to the age of forty years it is a fair inference that they have not been formed. The writer has seen an upper third molar erupting at the age of sixty. The cases of suppressed teeth next in point of frequency are those of the bicuspid teeth. If, however, the corresponding teeth are all present in the dental arch, a well-founded suspicion of impaction of the missing tooth may be entertained.

The extreme of suppressed formation is represented in a case described by Guilford.[1] A patient over fifty years old had never erupted any teeth, deciduous or permanent; the alveolar arches revealed no evidences of enclosed teeth, but had the appearance of typical edentulous jaws; the alveolar bone itself was primitive. The case appeared to be

[1] *American System of Dentistry*, vol. iii.

sporadically hereditary, a grandparent and an uncle exhibiting a like condition. The cases are interesting also because of additional evidences of faulty evolution of dermoid structures. In the first case cited no sudoriparous glands appear to have formed, and there was but a faint growth of hair on the cranium, and none on the face and body. The uncle was hairless and edentulous from birth. Guilford found in other members of the family an absence of the full complement of teeth.

Excess.—The possible occurrence of a condition in some respects the reverse of the preceding has been much written of and discussed—*i. e.*, the occurrence of a complete third denture. There can be but one conclusion from an examination of all the evidence thus far presented, and that is that no clear and well-authenticated cases are made out. Isolated cases of the appearance of teeth subsequent to the loss of all of the second denture are not infrequent; and, so far as clear records can be obtained, are resolvable into cases of the eruption of supernumerary teeth. While these cases are, at least for the present, to be held as unproved in connection with elderly persons, a well-authenticated case of multiple dentition in a child is recorded by Catching.[1] Between the sixth and the seventh month the eruption of one set of teeth was complete; within three months all of these had been lost. Between the eleventh and fifteenth months another period of dentition occurred, the teeth of this second denture being of such faulty structure as to crumble away quickly. At the age of two and one-half years a third dentition appeared, which caused the child such inconvenience that the teeth were extracted by the mother. At the age of eleven years a fourth series erupted, incomplete through the absence of six teeth. At the age of fifteen these teeth were sound and firm.

Fourth Molar.—The molar series of man, particularly in the lower negroid races, may consist of four instead of three members. When the fourth molar appears in the white races it is usually as a stunted member, a conical or peg-like tooth, similar to that which occasionally replaces the third molar. There is rarely room posterior to the distal wall of the third molar for their eruption, so that they make their appearance in the region shown in the illustration. S. M. Hartman,[2] L. D. S., of Victoria, B. C., has furnished the model (Fig. 191) of a case where the molar form of the fourth tooth is unusually well pronounced.

Supernumerary Teeth.—Any teeth in excess of the normal thirty-two, although clearly cases of reversion of type in many instances,[3] are included in the category of supernumerary teeth. Super-

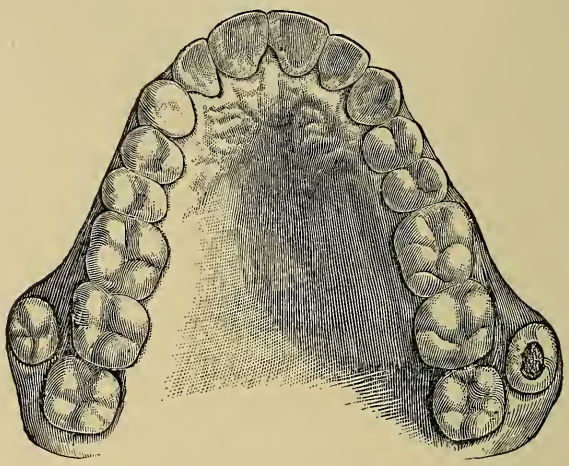
[1] *Southern Dental Journal*, Oct., 1886.

[2] *Dental Cosmos*, 1891.

[3] A. H. Thompson, *American System of Dentistry*, vol. iii., and Kirk's *Operative Dentistry*.

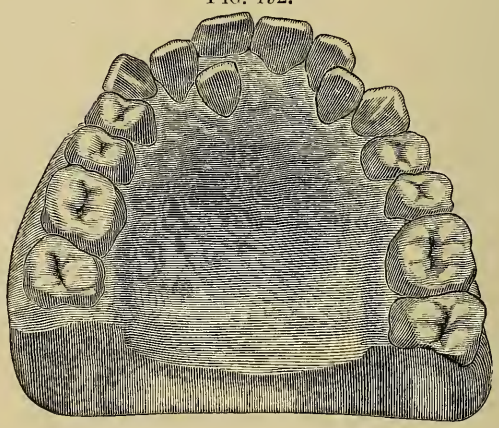
numerary teeth appear as simple unmodified cones, or as combinations of cones resembling the forms of teeth. The conical form is most common. Cases where these peg-like teeth appear around the third molars singly or in number are numerous. Their appearance in any

Fig. 191.



situation is evidence that the normal number of dental cords has been exceeded. They are perhaps all to be regarded as cases of long reversion, not alone because they increase the number of the dental series,

Fig. 192.



but because they have primitive forms, a modification of the forms found among the reptiles and fishes.

Guilford[1] divides supernumerary teeth into those having typical anatomical forms and those having the conical form.

[1] *American System of Dentistry*, vol. iii.

Supernumerary incisors in either jaw having typical forms are not uncommon. In the upper jaw supernumerary centrals and laterals both appear, the latter more frequently (Fig. 192). Supernumerary teeth may occupy any position relative to the dental arch, but are more frequently seen at its lingual side. The compound cone occasionally appears (Fig. 193). In addition to molars and incisors, supernumerary bicuspids are occasionally found; supernumerary cuspids are very rare.

Fig. 193.

Unless supernumerary teeth are a source of offence either through their positions or appearance, they need not be disturbed. If they are found to be so, they may be extracted.

Malpositions of the Teeth.

A tooth is said to be in malposition when it is not in normal relation with the dental arch to which it belongs and to its antagonizing teeth of the opposing arch. Teeth are found in abnormal positions as the result of a variety of causes. Some of these operate prior to, during, or immediately after eruption; some long after the eruption of the teeth, and some because of non-eruption.

Malpositions which are remediable through the application of mechanical force applied by means of suitable apparatus belong to operative dentistry, as has been stated. They are fully treated of in works upon operative dentistry [1] and orthodontia,[2] so that their discussion in a treatise upon pathology might seem a work of supererogation; the plan of the book, however, demands their brief mention.

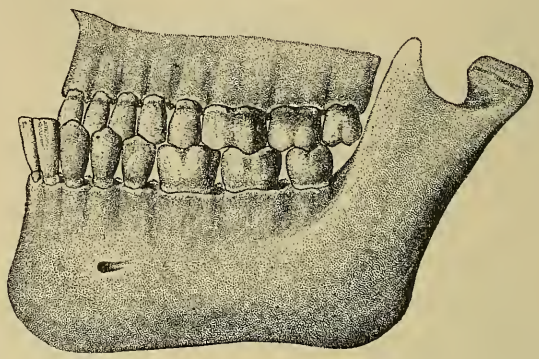
Malposed teeth may occupy any position relative to the dental arch, and any teeth of the dental series may be the offenders, although most commonly noted in connection with the incisors. So common is some degree of irregularity of the position of the lower incisors that its appearance is scarcely regarded as abnormal. The teeth may be inside or outside the dental arch, or have their transverse axes at any angle with the arch line—*i. e.*, may be rotated in any manner. In the most aggravated cases an entire half denture may be malposed as regards its relations with the opposing or antagonizing half. Instead of having the upper teeth occluding outside the lower, they may occlude inside (Fig. 194). They may occlude squarely without incisor overlapping. Both of these abnormal conditions are, of course, due to lack of correspondence between the development of the lower and upper jaws. If one jaw has developed normally, the other has necessarily developed insufficiently or too much.

[1] *American Text-book of Operative Dentistry.*

[2] Guilford, *Orthodontia;* Angle and others.

Malpositions of entire groups of teeth are found attended by an insufficient development of the alveolar bone of that region. They may also be caused by an excessive development of some section of the alveolar bone. The underlying causes of these gross aberrations are

Fig. 194.



only imperfectly made out. The reasoning adopted in discussing their causes and the conclusions reached appear to be largely speculative, although some of them are plausible. (The reader is referred to monographs upon orthodontia for the full discussion of these matters.)

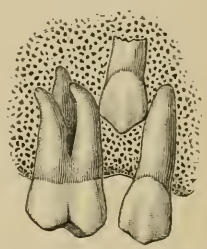
CAUSES OF MALPOSITIONS.

The causes for the malpositions of individual teeth are frequently traceable with a reasonable degree of certainty. The upper lateral incisor has an inherent disposition to erupt inside the dental arch; its crown during the formative stage lies slightly behind the crowns of the cuspid and central incisor; again, the forms of the alveoli of the temporary teeth, if regarded as rounded triangles on section, have the bases of the triangles of cuspid and central incisor outward, while the base of the triangle of the lateral incisor is inward, hence a line inward to the arch is the direction the crown of the lateral incisor tends to follow. Erupting normally, this tooth has a disposition to cause outward displacement of the cuspid. The lower incisors, held in an arch by the nature of the occlusion of the upper teeth, and no doubt also by the tongue, have not the same freedom of alteration of position as have the upper teeth; hence when the larger permanent teeth replace the smaller deciduous teeth they are crowded in the same arch-space and malposition results. It is evident that comparatively slight forces may deflect the direction of eruption of teeth, as they are only partially formed at the eruptive period, and are loosely enclosed.

Effects of Extraction of Deciduous Teeth.—The effects of the extraction of the deciduous teeth largely depend upon the time at which

the extraction is done. The general effect of extraction of the temporary teeth before their permanent successors are ready or nearly ready to occupy their places is a lack of space for the accommodation of the permanent successor, causing a delay in its eruption. The extraction of a temporary tooth interferes not only with those formative changes in the alveolar bone which afford increased space for the succeeding permanent tooth, but interferes also with the resorptive process which frees the permanent tooth from the roof of its cavity. An additional feature is the usual narrowing of the space from which the tooth has been removed. These conditions are more clearly observable in the case of the too early extraction of the temporary second molars. Not infrequently these teeth are extracted prior to the seventh year, or even earlier. Four years or thereabouts must then elapse before the permanent successor makes its appearance. The crown of the latter lies in the base of the alveolar bone, covered upon all sides by bony walls, and its position is lower than the roots of the adjoining teeth, the temporary first and permanent first molar. The normal tendency of the latter tooth is forward, and in the absence of the second molar it may attain a position immediately contiguous to the posterior surface of the first temporary molar. If the extraction occurs before the eruption of the permanent first molar, the condition described is almost certain to obtain, hence when the period of eruption for the second bicuspid arrives, the tooth is compelled to take a direction inward or outward of the dental arch, or, as happens in some cases, the tooth does not erupt at all, but remains impacted or encysted. Similar effects may be noted in connection with the remaining anterior teeth. The injury caused by extraction has been said to interfere with the normal formative processes occurring in the follicle of the corresponding permanent tooth. Such an effect is not at all improbable.

FIG. 195.



Effects of the premature loss of a deciduous second molar.

Effects of Delayed Loss of Deciduous Teeth.—If the resorption of the roots of the temporary teeth does not keep pace with the advance of the permanent teeth, more or less deflection of the course of the latter is almost certain to ensue. Recognizing the positions of the crowns of erupting anterior permanent teeth in relation with the roots of the anterior temporary teeth, it is evident that the general tendency of faulty eruption in these cases is inward. Comparatively and actually slight forces may deflect the course of an erupting tooth, hence the lower incisors, erupting before the upper, even though inward of the arch, are frequently driven into the arch-line by the muscular force of the tongue; the upper incisors, erupting later and inward, are imprisoned

by the lower incisors. It needs but the contact of slight occlusion to transform a slight into a marked malposition.

A curious relationship is sometimes established through the long retention of a temporary molar: the alveolar process develops normally and carries with it bodily the retained temporary tooth to a higher level, to the same level occupied by adjoining teeth, so that the tooth is in correct occlusion, although raised far beyond its original level. The eruptive impulse of the permanent successor of such retained temporary teeth seems to exhaust itself without avail, and the temporary tooth remains as a permanent feature of the adult denture.

TREATMENT.—When it is evident that the presence of a temporary tooth has deflected the line of eruption of its permanent successor it should be removed. When temporary teeth are retained beyond the normal period of eruption of their successors some operators advise that they should be extracted, a procedure said by them to hasten the appearance of the permanent successors. This rule is too sweeping, for eruption may be delayed from a variety of but partially understood causes, and the violence of extraction may disturb instead of aid eruption. However, when there is a pulpless temporary tooth in the arch, and all of the corresponding permanent teeth are in position, the indication is to extract the pulpless tooth. The process of resorption is faulty and incomplete in such cases, and it is probable that the extraction of the tooth removes a mechanical obstruction to the eruption of its successor. In the absence of an appearance of the crown of a permanent tooth, and with no evidences of loosening of the healthy temporary tooth, the forcible extraction of the latter is rarely advisable. It has happened that firm and sightly temporary teeth have been extracted, but no permanent successor appeared.

FIG. 196.

Effects of premature loss of permanent first molars.

Effects of the Early Extraction of the Permanent First Molar.—The permanent first molar erupted from the sixth to the eighth year may suffer from dental caries, pulp-necrosis, or alveolar abscess before the tenth or eleventh year. If these teeth are extracted, a characteristic dental deformity follows. Recalling that the development of the jaw proceeds in such manner that the depth of the alveolar bone is constantly increasing until adult age, and that this development is only marked during and after the appearance of the second dentition, it is evident that the extraction of the permanent first molar is followed by a lessening or cessation of development of the bone adjacent; the formative process will not then deepen the lateral alveolar walls until the period of eruption of the bicuspids, not for three years or more. In the mean-

time the permanent incisors erupt and the alveolar bone deepens around them; their occlusion is not limited in extent by a corresponding deepening of the posterior alveolar bone. The lower incisors, being without check to their action, come to strike the upper incisors at their cervico-palatine portions; these latter teeth are gradually driven from their vertical positions until they assume almost a horizontal direction.

Treatment.—The treatment of this condition is preventative: the permanent first molars should not be extracted until the bicuspids have fully erupted, provided that their retention is possible. The permanent first molars, far from being, as was once held, the most worthless teeth of the dental series, are the most important. Their eruption at an early period is a distinct indication of the important influence their presence exerts upon the normal development of the alveolar bone about and posterior to them. Their eruption is about synchronous with the completion of the evolution of the alimentary canal and its appendages; hence their office in the increased mastication, normal at this time, is clear; their presence beyond a doubt determines the extent of the formative process which shall occur in the alveolar bone posterior to them. From the period of their eruption they should be carefully scrutinized; and even though caries and subsequent disease-processes act to the extent of alveolar abscess, the conditions are to be vigorously treated, so that the teeth may be retained until after the eruption of the second bicuspids.

IMPACTED AND ENCYSTED TEETH.

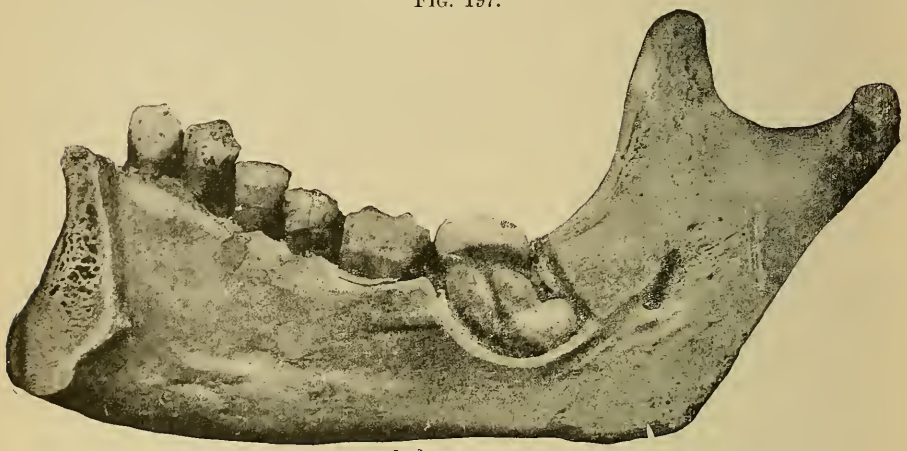
The extreme extent of dental malposition is reached when the permanent teeth do not erupt at all. Instead of presenting in the dental arch, they may be entirely imbedded in the substance of the bone, either remaining there, with or without pathological manifestations, or erupting in some very unusual situation. In other cases a distinct cystic tumor forms about the enclosed tooth.

Impacted Lower Third Molars.—By far the most common dental impaction is that of the lower third molar. The extent of impaction varies from a partial eruption, or partial imprisonment of the tooth by its bony surroundings, to its entire imprisonment in any part of the maxilla. Many of the more severe cases treated of under the head of difficult eruption, if unrelieved would be included in the category of impacted teeth.

In Fig. 197 is shown a lower third molar presenting the effects of a previous impaction. The irritation caused by the efforts of the tooth to disengage itself or to overcome the resistance to its eruption has caused an active formative reaction in the pericementum, resulting in a hypertrophy of the cementum.

If the distance between the posterior surface of the second molar and the columns of the coronoid process be very short, it is evident that upward eruption is impossible, so that the tooth may assume any direction of movement, the most common being forward, the axis of the tooth

FIG. 197.



Right half of lower jaw, showing an impacted third molar. (Cryer.)

FIG. 198.

Inner side of left half of same lower jaw. (Cryer.)

changing its position until the tooth may lie in a horizontal position or even become inverted.

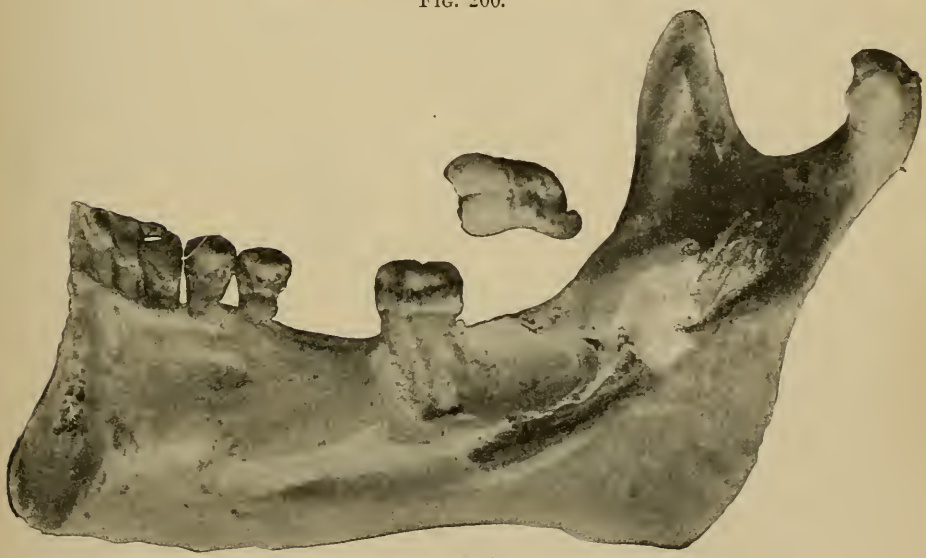
Fig. 198 is taken from the same jaw as Fig. 197, but shows the opposite side; the impaction is pronounced. Fig. 199 shows another

case with different anatomical surroundings. In the first case there were evidences both in the tooth, in its bony surroundings, and in the external cortical bone, of the results of the irritation produced by the efforts at eruption. The cementum was thickened; the outer follicular wall,

FIG. 199.

(Cryer.)

FIG. 200.

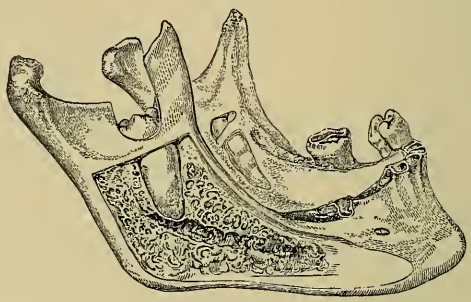


(Cryer.)

the tissue designed to form the alveolar periosteum, had exercised its formative osteogenetic function, and a capsule of bone had formed about the tooth; it lay in a bony chamber. The pressure exerted upon the distal wall of the second molar had resulted in a pressure-resorption of its root until the pulp-chamber was encroached upon. In Fig. 199 the

root-development has caused impingement of the root-apex upon the inferior dental canal. These were both post-mortem cases, and no records of their clinical histories were obtainable. The symptoms produced could only be surmised by the nature of the anatomical relations and the pathological evidences. There may have been a prolonged but mild periostitis, probably a continued pulp-irritation; and in the last, neuralgia of any grade of severity.

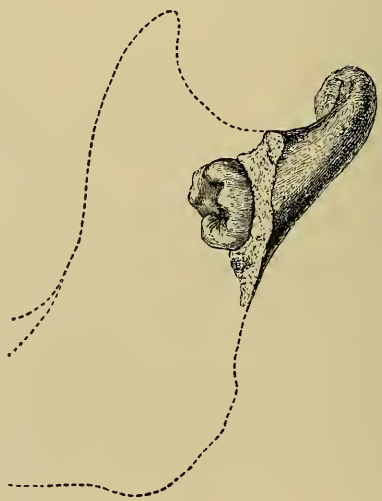
Fig. 201.



Wisdom-teeth imbedded in the rami of the lower jaw. (Tomes.)

Judging from post-mortem records, cases of impacted third molars are more common than generally believed. Instead of remaining in

Fig. 202.

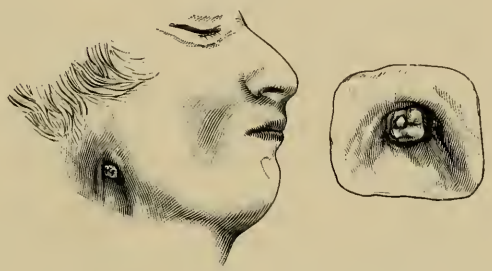


Wisdom-tooth buried in the ramus. (Tomes, after Marshall.)

the alveolar portion of the bone, the impacted tooth may come to occupy a cavity in some portion of the body or the ramus of the bone (Figs. 201 and 202). The positions of the teeth in such cases tend to

confirm Tomes' theory of the development of the jaw. The jaw being lengthened, and the ramus developing through conjoined deposition and resorption of bone, the crown of the tooth appears to be either fixed in a bony nucleus and transported to some distant point in the developmental progress of the jaw, or to be irregularly shifted about during jaw-growth. At later periods the pressure exercised by root-formation disturbs the relations of the tooth with its earlier surroundings. These efforts at eruption may at late periods cause the appearance of the tooth in odd situations (Fig. 203). The crown of the tooth in this

Fig. 203.

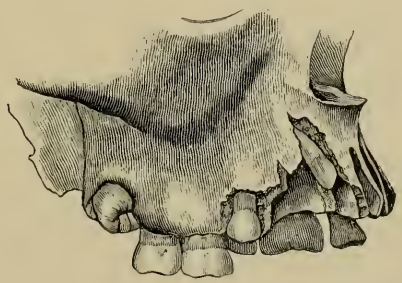


From a wax model in the museum of the London Odontological Society. (Tomes.)

case made its way through the angle of the bone and through the muscles and skin. The opening in the skin healed upon extraction of the tooth.

Impacted Upper Third Molars.—Some grades of impaction of this tooth have been spoken of under the head of difficult dentition. The

Fig. 204.



Upper jaw, with the third molar directed forward, and impinging upon the second molar. The small tooth situated high up in the anterior part of the jaw, was forced there by the spade of the grave-digger. The artist's accuracy in delineating all parts of the specimen has rendered this explanation necessary. (Tomes.)

most common is imprisonment of the tooth and its subsequent partial eruption in a horizontal position, the crown pointing toward the cheek (Fig. 204). The crown of this tooth may in rare cases be directed

inward or backward, in the latter case being arrested by the pterygoid plates of the sphenoid bone.

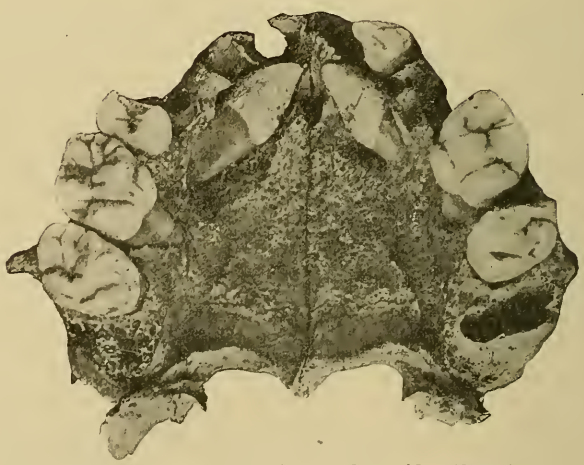
In a case recorded by Tomes (Fig. 205) the extraction of the second molar revealed the third molar in a reversed position, its roots occupying the depression between the roots of the second molar.

FIG. 205.

A second molar of the upper jaw, with the wisdom-tooth inverted and embraced within the roots. (Tomes.)

Impacted Cuspids.—In point of frequency of impaction the upper cuspids stand next to the lower third molars. It will be recalled that the upper cuspids lie high up; the floors of their crypts, in which they lie *loosely*, are at a higher level than those of the adjoining teeth; they erupt at a much later period, and their crowns, as with the other anterior teeth, lie inside the roots of their predecessors. All of these are elements which might cause displacement of the developing cuspids. Should the advance of eruption not keep pace with the development of the alveolar bone, imprisonment is likely; again, the dense bone immediately about the first bicuspid and lateral incisor may offer a deflecting resistance. Examining the texture of the bone about these parts, it is evident that the least resistance to the advance of a much-deflected crown is into the cancellated bone of the incisor portion of the alveolar process;

FIG. 206.



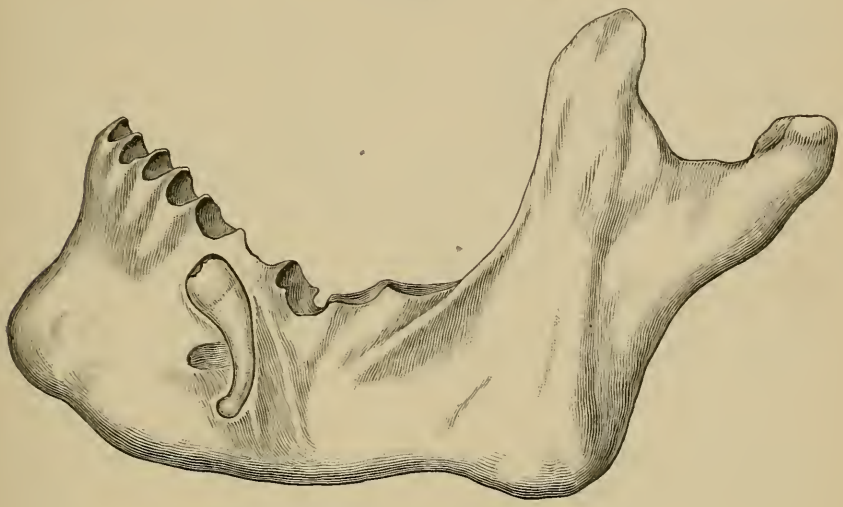
Abnormal jaw, showing impacted cuspids. (Cryer.)

hence it is most usual to find the crowns of these teeth lying with their cusp-point forward (Fig. 206). Several recorded cases have the positions shown; one or both of the teeth may be encysted.

Impaction of Other Teeth.—While impactions are most common

in connection with the teeth named, any other teeth of a denture may be imprisoned. Fig. 207 shows an imprisoned bicuspid whose root-development has been normal as regards its length, but whose

Fig. 207.

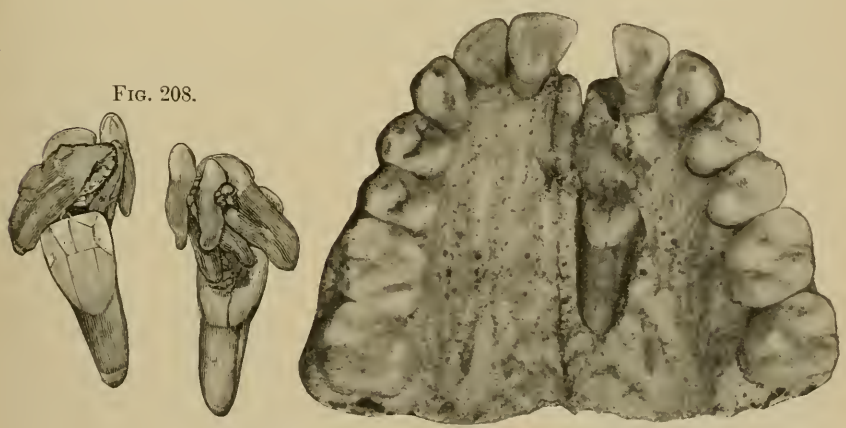


Impacted bicuspid. (Salter.)

curve has been modified by the resistance of surrounding tissues. Figs. 208 and 209 exhibit an impacted central incisor, whose retention was,

Fig. 209.

Fig. 208.

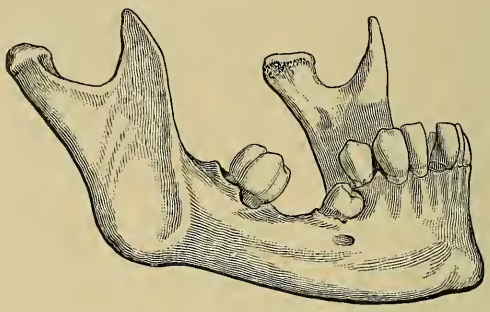


Imprisoned central incisor. (Kirk and Cryer.)

no doubt, determined and malposition caused by the development and presence of the brood of supernumerary teeth which surround its crown.

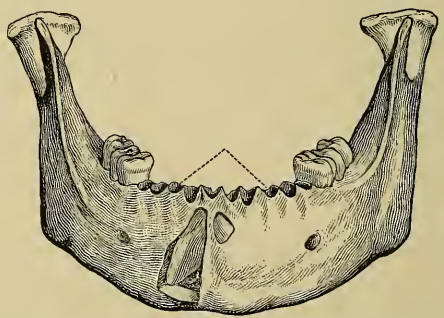
Symptoms.—The most common symptom attendant upon impaction of teeth, judging from the obtainable records of cases, is trifacial neuralgia of any degree, caused by impingement of the malposed tooth

Fig. 210.



Lower maxilla, in which the right second bicuspid is placed obliquely, the root being directed backward. The crown, though exposed, does not rise above the level of the alveolar margin. (Tomes.)

Fig. 211.



Maxilla, in which the temporary cuspids (the sockets of which are shown by the dotted lines) were retained, and the permanent canines developed within the substance of the jaw. The bone has been removed on the one side to show the direction taken by the tooth, which has been twisted on its axis to the extent of a quarter of a turn. (Tomes.)

upon nerve-filaments or trunks. Cryer[1] records a case where a supramaxillary neuralgia was traced to the presence of a central and lateral incisor, and a cuspid tooth in the anterior wall of the antrum; they were only discovered by an exploratory operation (Fig. 212). A cure of the neuralgia was effected by their removal.

Fig. 212.

Impacted third molars frequently give rise to heavy rheumatic pains about the side of the face and jaws, and no doubt in such cases as depicted in Fig. 199 would cause intractable and diffuse maxillary neuralgia. Salter[2] records a case of long-standing and intractable neuralgia,

[1] *Dental Cosmos*, 1896.

[2] *Dental Pathology.*

exhibiting a constant painful area upon the scalp, and in which heat and tenderness were noticed over a swelling upon the hard palate. Immediate and permanent cessation of the neuralgia followed removal of the teeth.

Symptoms of maxillary periostitis — heavy, gnawing, and dull, throbbing pain, with more or less heat and engorgement of tissues — are noted as an accompaniment of impacted teeth. Such symptoms may herald the appearance of the tip of the tooth through its bony covering and gum.

Cases of maxillary abscess, in the absence of their usual (dental) cause, may run a prolonged and painful course,[1] involving neighboring structures, and after free venting be found to have arisen about an impacted tooth.

Occasionally a circumscribed swelling is noted upon some aspect of a jaw, most frequently upon the palatal portion of the superior maxilla, which is attended by inflammatory symptoms, and an incision reveals an impacted tooth.

Quickly forming cysts of the jaw upon receiving surgical treatment may be found to contain the crown of an entire tooth, this evidently being the centre of irritation from which the cystic formation had its origin.

Diagnosis.—The first point of observance in cases of suspected tooth-encystment is an examination of the dental arches. Are all of the permanent teeth in position? Given the absence of, particularly, a lower third molar from the dental arch, with a history of no eruption, and a persistent neuralgia, particularly if occasionally accompanied or alternated by heavy rheumatic, what are known as bone-pains, and finding no other evident cause of the neuralgia, the effects of an impacted tooth would be naturally diagnosed as the source of the disturbance. Impacted teeth which lie horizontally or nearly so along the palatal vault frequently cause a swelling. This, taken in conjunction with the absence of a tooth from the dental arch, points to a diagnosis of impaction.

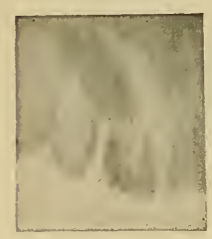
In very many cases of impaction diagnosis has been a mere accident, discovery being made in the course of an exploratory surgical operation. Modern science solves with the *x*-ray the difficulties attendant upon the diagnosis of impacted teeth. B. H. Catching[2] was the first to practically apply this diagnostic test for the location of an impacted tooth. The left upper central incisor of a female aged nineteen became loosened, and an exploration through its pulp-chamber revealed a hard body occupying a position part way up the root, which had undergone

[1] See Garretson's *Oral Surgery*, and Salter's *Dental Pathology*.

[2] Catching's *Compend*, 1896.

resorption to that point. The cuspid of the left side was absent from the arch. A skiagraph of the parts (Fig. 213) revealed the missing cuspid, whose crown had impinged upon and caused resorption of the root of the central incisor.

Fig. 213.

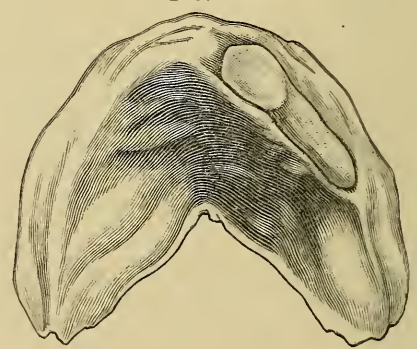


X-ray photograph, showing the malposed cuspid entirely embedded in the bone, and pointing on the central.

Impacted teeth may become uncovered at some aspect late in life and the condition be discovered incidentally. Cases are recorded where the pressure of a plate has caused the resorption of tissues overlying an impacted tooth, thus revealing its presence. Fig. 214 illustrates a case where the presence of an impacted cuspid was revealed at the age of seventy years, through resorption of the alveolar bone and the gum-tissue covering the tooth.

Beyond a doubt, the *x*-ray will be generally used in the future to determine the position of permanent teeth absent from the dental arch; and will be used as a means of diagnosis when the presence of impacted teeth or an odontoma may be suspected.

Fig. 214.



Treatment.—The treatment of cases of impaction is the removal of the offending tooth. Whether or not this comes within the province of the dental operator depends upon the position of the tooth, and, incidentally, upon the usual range of practice of that particular practitioner. When the tooth is imbedded deep in the substance of the jaw, access to it involves the etherization of the patient and the removal of the bone which obstructs the path of extraction; this may be an operation of some magnitude, and is usually done by a special surgical practitioner. When, however, it is evident that the obstructions to the removal of the tooth consist of the soft tissues and but a lamina of bone, the operation for removal is clearly within the

province of the dental operator. For example, the presence of an impacted cuspid is determined lying horizontally along the lateral aspect of the roof of the mouth. The parts may be injected with a cocain or eucain solution, and a curved cut made with a sharp bistoury through the soft tissues at the dental side of the swelling to the bone. The flap thus outlined is raised from the bone, the flap including the periosteum. A large sharp bur is then employed to remove the covering bone. When the tooth is freely exposed it may be dislodged with forceps or elevator. The parts are then washed with a hydrogen dioxid solution, dried, the flap pressed back into place, and steresol (which see) painted over the parts.

The treatment of such cases as Fig. 207 is the same.

SECTION III.

AFFECTIONS OF ENAMEL AND DENTIN.

CHAPTER XI.

AFFECTIONS OF THE ENAMEL.

It has been repeatedly stated in the preceding pages that enamel plays an entirely passive part in disease-processes. Being cut off at the completion of its formation from all sources of nutrition, it is deprived of all defensive mechanism against agencies which may threaten its destruction. Furthermore, recognizing this absence of nutritive mechanism, it is evident that enamel cannot be subject to the classes of diseases which affect vital tissues; that is, degenerations and the causes of degenerations have no representatives in affections of the enamel. While the enamel may suffer loss of substance, it is not possible that constructive or retrogressive metamorphosis can take place.

The enamel can be regarded only as an inert chemical substance, the exact chemical morphology and composition of which are but imperfectly understood. It is acted upon by a variety of physical forces and chemical agencies which threaten the destruction of its substance, either through mechanical injury or chemical solution, and against which agencies it is incapable of any but mechanical and an unalterable chemical resistance. The disease-causes operating against the integrity of the enamel of the teeth are mechanical and chemical; both of these cause a loss of substance, but in a different manner; again, they may act together. The mechanical forces act as destructive agents, either by removing the enamel particle by particle by a process of abrasion, or by causing fracture, complete or incomplete, of masses of enamel. It appears in some cases that, in addition to direct mechanical violence, abnormal temperatures to which the enamel may be subjected cause linear fractures of enamel-plates. Enamel suffers from chemical solution both through the action of acids formed in the mouth during fermentative processes, and probably, by morbid acid secretions of glands, also through the action of acids present in food or taken as medicinal agents.

Mechanical Injury of Enamel.

It was pointed out in Chapter VIII. that the resistance of enamel against crushing-forces is comparatively slight, and that its resistance in this direction is much modified according or not as the tissue is uniformly and firmly supported by dentin. Masses of enamel, therefore, which are deprived of the normal support of underlying dentin are in danger of fracture. It is frequently noted that teeth which give no very apparent external indication of loss of dentin, the enamel contour being almost intact, may exhibit an extensive disorganization of the dentin, through a comparatively light stress causing fracture of the enamel. Biting upon a crust may be followed by crushing of the greater portion of the enamel-cap, revealing, as the underlying cause of the fracture, disappearance of dentin support.

The enamel of teeth appears to differ in fragility, both as regards individuals and the several teeth of one denture.

Fracture of enamel *en masse* occurs more readily with pulpless teeth, those in which decomposition of the contents of the dentinal tubuli has taken place, than it does with teeth containing vital pulps. Its resistance is much greater in teeth in which the tooth-pulp has been devitalized and removed and the canals filled under aseptic precautions, with absolute exclusion of saliva, than when the saliva has been permitted free entry into the interiors of the teeth.

Through either lack of perfect adaptation or improper character of support, enamel-walls underlaid by gold or amalgam fillings are much more liable to fracture than if supported by zinc-phosphate cement. It should be remarked in this connection that the chief and most valuable use of zinc phosphate in dental practice is as a mechanical substitute for lost dentin.

The accepted rule of operative dentistry, not to pack gold against unsupported enamel-walls, is a well-founded one. The danger of fracture is twofold: first, fracture during the operation of impaction; secondly, subsequent fracture due to improper support.

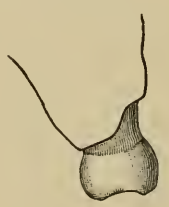
Enamel (see Chapter VIII.) has lines of least mechanical resistance, which are in planes at right angles to the general direction of the enamel-rods; that is, enamel fractures most readily along the lines of the cementing-substance of the enamel. In addition to these uniform lines of low resistance there appear to be others which invite fracture along straight lines passing from the occlusal edges of the enamel to its cervical border; longitudinal fracture of the entire length of an enamel plate occurs. These lines may be observed in the enamel of persons who habitually break ice and other hard substances with their teeth. Identical lines of fracture are found in enamel which patients deny having

subjected to such usage; they have been attributed to the contact of excessively hot or excessively cold substances with the enamel, a reasonable hypothesis.

ABRASION OF THE ENAMEL.

By abrasion of the enamel is meant a wearing away of its substance through the friction of mastication when gritty substances are present. Some forms of abrasion have been attributed to the too vigorous use of hard tooth-brushes, particularly when gritty tooth-powders are employed. There is no doubt that mechanical abrasion about the necks of teeth is produced in this manner, the gum-line receding beyond the enamel-border, exposing the cementum: but a careful examination will reveal the cementum, and next the underlying dentin to be affected; the enamel is not abraded. These tooth-brush abrasions are quite characteristic (Fig. 215). In well-kept dentures the gums are seen to have receded from their normal line, but exhibit no evidences of turgescence; the roots of the teeth, upper and lower, are exposed to a greater or less extent along their labial and buccal, but not along their lingual aspects; and they are excavated to variable depths, upon the bicuspids and first molars more than upon the other teeth, as here the greatest force of brushing is received. The depressions have a normal dentin color, which in smokers may be periodically blackened by deposits of carbon. If caries supervenes, the abraded areas lose their normal color, and may be readily indented by sharp instruments, which they resist before the advent of caries. The bicuspids and molars particularly may be grooved in such manner as to require restoration by fillings.

Fig. 215.



Abrasion of enamel-surfaces through the constant rubbing of metallic clasps of prosthetic appliances has been said to occur, but it is impossible to disassociate in this connection the effects of the acids of fermentation. Food-débris on the inner surface of a clasp undergoes fermentative changes, and acids are formed which act as decalcifying agents upon the enamel; so that the abrasion of the clasp is not upon normal enamel-surfaces, but upon partially decalcified enamel. This fact becomes more evident when it is observed that the abraded surfaces are not smooth and polished, but are roughened. There is no doubt that the continued rubbing of a clasp upon a tooth will abrade enamel slightly, but the surface produced is polished. The abrading effect of clasps in contact with cementum or

Fig. 216.

dentin is unquestionable, but there is also in such cases the conjoined effect of acids of fermentation.

By far the most common cause of the abrasions noted upon the occlusal surfaces of teeth is tobacco-chewing. The silex contained in tobacco-leaf acts as an irresistible abrading agent. The cases in which this form of abrasion is most marked are those in which there has been originally but little overbite of the upper teeth, or in which the teeth have a tip-to-tip occlusion. There appears to be a tendency with this type of occlusion for the occlusal surfaces of the teeth to abrade. There are always a free lateral movement of the mandible, and a type of surface-contact resembling that of the herbivora. Whether due to the type of occlusion itself or that with this type of occlusion the individual more frequently chews the siliceous stems of vegetables, it is with such patients that general abrasion is most common. It is also frequent in those cases presenting the first degree of prognathism. In all of these cases, after the abrasion has worn down the teeth to the cusp-bases, the occlusion tends to become of the tip-to-tip variety. When there is a more marked overbite occlusion, with a consequent lessening of lateral movement of the mandible, the teeth do not acquire flattened contact-surfaces, but their cusps increase in sharpness and pointedness.

The effects of abrasion are to leave sharp enamel-edges standing, which when the dentin becomes exposed grow more sharp and more pointed. Being without direct dentin support and receiving stress in abnormal directions, the enamel tends to fracture.

In the overbite cases the conjoined action of abrasion and of gradual molecular fracture of enamel creates curiosities of occlusion. The teeth of the upper and lower jaws may interlock their cusps in such a manner that the line of junction is almost invisible.

The sharp enamel-edges produced not only increase the liability to enamel-fracture, but act as constant sources of irritation to soft tissues which come in contact with them, the most frequent result being abrasions upon the side of the tongue, producing ulcers of a sometimes chronic type which acquire indurated edges and simulate syphilitic sores or epithelioma. The causal relationship between sharp edges of the teeth and lingual epithelioma appears to be quite clear in some cases.[1]

Sores which have given evidence of malignancy and been diagnosed as malignant growths have been cured by rounding and polishing sharp and irritating enamel-edges of teeth.

Treatment of Abrasion.—If seen early enough, the treatment of abrasion should be preventative. Abrasion has unquestionably been arrested by cessation of the tobacco-chewing habit. A difficult class of cases to treat is found in those highly nervous individuals who

[1] Garretson's *Oral Surgery*.

grit their teeth during sleep. It is probable and reasonable that this cause alone may serve to explain abrasions traceable to no other source. The cure of such cases as these could only be possible through the wearing at night of some modified form of interdental splint. The cases naturally indicate the medicinal use of a bromid before retiring.

It is rare that any remedial measures are adopted or, indeed, even advisable, unless the abrasion wears the teeth down far below the cusp-line, although the dentin exposed through the loss of enamel may become hypersensitive and require treatment. The mildest agent affording relief is carbolic acid. For the relief of periodical hypersensitivity the use of a mouth-wash of dilute phénol sodique is effective if continued. Some spots of hypersensitiveness resist every measure except applications of strong mineral acid. The action of these acids, sulfuric, nitric, or hydrochloric, being chemically destructive, the spots of application must be subsequently excavated and filled.

The radical relief of the condition is by the operation called "shoeing" the teeth. Each abraded tooth is to have built upon it sufficient gold-foil to receive the stress of mastication. The fillings must be of such thickness that the stress of mastication cannot alter their forms, and be built as solidly as molten gold; to assure this they are made of heavy rolled foil, No. 30, No. 60, and for the final surfaces No. 120. In many cases it is advisable to use for this purpose platinum-gold foil, which resists attrition better than gold-foil itself.

Cases of attrition of the oral teeth and of the bicuspids are frequently seen where the loss of several molar teeth has permitted the entire stress of mastication to come upon the anterior teeth. These cases are treated by adjusting bridge fixtures in appropriate spaces, whose occlusion shall raise the bite—*i. e.*, separate the jaws to the proper extent; hard-foil fillings are then inserted in the worn anterior teeth, restoring to them their original forms.

In case the abrasion is very deep upon all of the teeth, it is advisable to fit gold crowns of the barrel variety upon the posterior teeth and adjust porcelain-faced crowns over the anterior teeth. The pulps of the teeth need not necessarily be destroyed.[1]

Erosions of the Teeth.

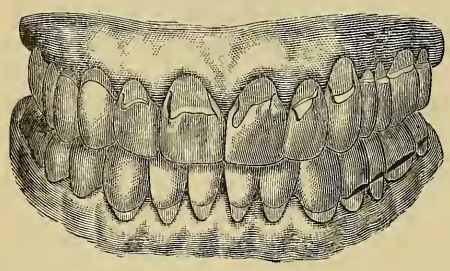
Erosion of the teeth is a term applied to the decalcification of the hard tissues of teeth in such a manner that broad, shallow excavations are made in the enamel, and in such situations that the acids of fermentation and mechanical abrasion are clearly excluded as exciting causes. The excavations occur upon surfaces where fermentative processes are

[1] See *American Text-book of Prosthetic Dentistry*, chapter xxii.

in least degree and where attrition is nil. Figs. 217, 218, and 219 illustrate the characteristic appearance of areas of erosion.

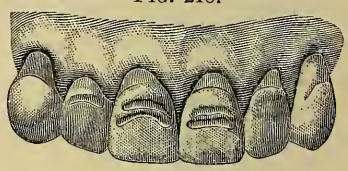
The labial faces of the anterior teeth are more frequently affected than those of any of the other teeth. These surfaces appear as though sections

FIG. 217.



(Darby.)

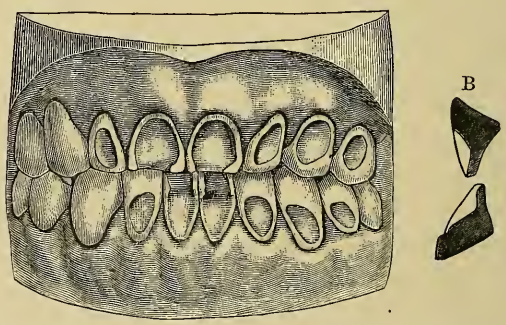
FIG. 218.



(Darby.)

had been bodily cut out of them. The enamel is affected to a greater extent than the dentin, forming shallow excavations in the teeth. When

FIG. 219.



A case of erosion (drawn from the cast): B, silhoutte from a perpendicular line through the left centrals, upper and lower, showing the loss of substance. (Black.)

the destructive action lays bare the dentin, neither it nor the enamel presents any of the appearances of dental caries, the eroded surfaces being smooth and polished and of normal hardness.

CAUSES.

From the chemical composition of the enamel it is at once evident that the essential cause of the loss of structure must be an acid; only acid substances can dissolve it. The question of mechanical abrasion may be set aside because erosion has been found upon the teeth of individuals who had never used a tooth-brush, and the situation of the areas precluded the idea of abrasion by mastication. It is evident that the acid must be of localized formation, because if of general distribution the lingual and occlusal faces of the teeth would be affected

equally with the labial. It will be observed that the areas of erosion are in situations in which food-débris, fermentable material, collects in least amount, and where the acids of fermentation necessarily form in slight amount. This survey materially narrows the field of inquiry. What structures or substances can form acids or acid bodies about the labial faces of the teeth, excluding fermentative processes? The inquirer is of necessity driven to the belief that local acid secretion is the active cause. Secretion implies the existence of glandular tissue, and the only glands in close relationship with the labial faces of the teeth are the muciparous glands of the lips; mucus-forming glands, called labial follicles. Edward C. Kirk[1] first pointed out the probability of altered (acid) secretion of these glands being the active cause in the production of erosion.

It has been recorded by those who have made a special study of the condition that females are more frequently affected than males; that it rarely becomes evident before the age of thirty or later; and, finally, that patients in whom it is noted are usually the victims of the condition indefinitely known as the gouty diathesis. The observations of Kirk, Darby, Jack, and myself have all had a singular unanimity of agreement in this direction. Most of the patients give a family history of gout, and very commonly a personal or contemporary family history of rheumatoid arthritis or rheumatism. Even when the existence of rheumatoid or gouty affections is denied by both patient and medical attendant, it is rare that the patient does not complain of some general disorder, the usual ones being neuralgia of long standing, marked anæmia, or perhaps neurasthenia. Be the condition what it may, the essential disease-process is one which may be traced to the effects of suboxidation in the tissues.

The researches of general pathologists as to the products of cell-oxidation but insufficiently explain the origin and significance, both chemical and pathological, of a series of bodies related to urea, unquestionably waste-products resulting from broken-down, oxidized albuminous matter. These related products are uric acid, xanthin, and hypoxanthin, which represent, chemically at least, degrees of albuminous oxidation—urea the most oxidized, hypoxanthin the least. While the writer is aware[2] that pathological chemists refuse to recognize uric acid as a midway product in urea-formation, yet it cannot be denied that uric acid will form in increased amount if the oxidizing function be below par; all clinical and scientific reasoning appears to point to this end. Note the conditions in which an increase of uric-acid formation is

[1] *Dental Cosmos*, 1886.

[2] See Levison on *Uric Acid*; Luff, Croonian Lectures, 1897; Halliburton, *Chem. Physiol. and Pathology.*

observed. It should be remarked parenthetically that uric acid cannot exist free as uric acid in the blood, but when formed combines immediately with sodium or magnesium, doubtless displacing the negative radical of compounds, and forming sodium or magnesium quad-urates.

An excess of urates is formed whenever there is an excess of white blood-corpuscles, in conditions of anæmia, leukæmia, and so on. The significance of these conditions lies more in the deficiency of red corpuscles than in the excess of white. Less red corpuscles, less hæmoglobin, less oxygen carried, hence diminished oxidation in the cells. Again, in the opposite, the plethoric individual, who suffers from acute outbreaks of gout, large quantities of albuminous food are taken, amounts far in excess of that required to replace waste; frequently, owing to glandular disturbances of the alimentary canal, the material is insufficiently elaborated, and the tissues of the body are drenched with an excess of material in unfit chemical state for cell-metabolism; the amount of oxygen, actually large, relatively small, is insufficient to oxidize all of the material of which the cells of the body should rid themselves. Conjoined with this, the same individuals usually consume too much alcohol in one form or another, and alcohol notably lessens cell-oxidation.

The relation between an excessive formation of urates and deficient oxidation may in a like manner be made out in many of the other conditions. A deficiency of oxidation or in the supply of oxygen is, of course, accompanied by retention of CO_2 in cells, so that all of the waste-products of cells are but imperfectly removed. It should also be noted that the products of cell-waste are acid in reaction.

"If the orbicularis oris muscles be dissected from the mucous membrane of the lip, the labial glands may be observed; they are more numerous near the centre than at the extremities of the lip"[1]—*i. e.*, the greater number overlie the labial faces of the incisors toward the necks of these teeth. "These are small racemose glands, their ducts lined with low granular epithelium; in the alveoli the cells are larger and columnar and stain less readily with carmine." "Their secretion is composed of water, mucin, and inorganic salts, sodium phosphate predominating, which gives the fluid its alkalinity under normal conditions. In conditions of irritation and consequent hyperæmia the secretion becomes increased in amount and acid in reaction (Kirk). The nature of the acid is not clearly known. Brubaker[2] suggested that it might arise as follows:

In the condition known as the gouty diathesis there are evidences of widespread irritation; the presence of an excess of waste-products appears to be the cause of the irritation. In consequence of the irrita-

[1] Brubaker, *International Dental Journal*, Dec., 1894. [2] *Ibid.*

tion there is a vascular reaction about glands, as those under consideration, which results in increased secretion, and as the oxidizing function is imperfect the CO_2 accumulates. "Given upon one side, the vascular side, of the cells the readily diffusible and alkaline salt sodium phosphate, Na_2HPO_4, and in the cell an excess of CO_2, existing as H_2CO_3, or carbonic acid, when the sodium salt diffuses into the cell the following reaction occurs:

$$Na_2HPO_4 + H_2CO_3 = NaH_2PO_4 + NaHCO_3.$$[1]

That is, dihydrogen sodium phosphate (acid sodium phosphate) and acid sodium carbonate are formed. Dihydrogen sodium phosphate, being acid in reaction, is capable of effecting the decomposition of the calcium phosphate of the teeth after the following manner:

$$NaH_2PO_4 + Ca_3(PO_4)_2 = NaCaPO_4 + 2HCaPO_4.$$

"The acid sodium phosphate formed in the gland-cells and given off by them attacks the calcium salts of the enamel, as above expressed, a double decomposition occurring, sodium calcium phosphate and acid calcium phosphate being formed. The latter, acted upon by additional molecules of the dihydrogen sodium phosphate,

$$NaH_2PO_4 + 2CaHPO_4 = NaCaPO_4 + Ca(H_2PO_4)_2$$

and the freely soluble diacid calcic phosphate is formed. In this way the calcium phosphate of the teeth undergoes decomposition into, first, mono, and, second, the diacid calcic phosphate." Brubaker immersed a tooth for a week in a solution of acid sodium phosphate, subjecting it daily to tooth-brush friction, and at the end of that time spots and grooves resembling areas of erosion made their appearance.

While admitting the probability that dihydrogen sodium phosphate is the decalcifying agent, Kirk maintains[2] that the reaction between the hypothetical substance, carbonic acid, H_2CO_3, and sodium phosphate is a chemical impossibility, as the reaction given involves the production of acid sodium carbonate in the presence of acid sodium phosphate, which cannot occur.

He suggests that the probable origin of the acid sodium salt is from the reaction occurring between hydrogen disodium phosphate, Na_2HPO_4, and uric acid, $C_5H_4N_4O_3$, citing from Hammersten that uric acid, soluble in a warm solution of hydrogen disodium phosphate, HNa_2PO_4, in an

[1] It is to be understood that Brubaker advances this explanation hypothetically, not as an assured demonstration, as he says there are no present tests by which such small amounts of the acid substance can be detected.

[2] Private communication.

excess of the acid, a double decomposition or atomic interchange occurs as follows:

$$HNa_2PO_4 + C_5H_4N_4O_3 = H_2NaPO_4 + C_5H_3N_4O_3Na,$$

resulting in the formation of sodium biurate and the acid sodium phosphate, H_2NaPO_4, which may act as a decalcifying agent after the manner described by Brubaker.

The uric acid involved in the reaction is to be regarded as a consequence of faulty oxidation in the glandular tissues. The sodium biurate found in the reaction given above may act as an irritant and induce a continuation of the glandular affection.

Kirk has pointed out that the contents of the labial glands in cases of erosion gave an acid reaction—*i. e.*, reddened blue litmus paper—in all of the cases tested by him.

It appears to be almost self-evident that there must be some modifying factor causing the peculiar forms of the eroded areas, and that it must be an abrasive. In many of the cases where the erosions are in the form of transverse grooves or pits there is no doubt that the action of the tooth-brush upon the decalcified parts removes the latter; the areas of erosion may be oval, circular, or irregular patches; again, decalcification may appear to occur over the entire labial surfaces of teeth uniformly; moreover, as stated, erosions in grooves may occur upon the teeth of persons who do not use a tooth-brush. It is evident then that in these cases the mechanical factor must be sought in the muscular movements of the lips and tongue. The greatest effect of lip-movement would be upon the entire labial faces of the central incisors, as the maximum force of contact of lips with teeth is at and near the median line, when the lips are alternately raised and depressed. The action of the tongue upon the labial faces of the teeth would be in a curved line passing across the labial and buccal surfaces of the teeth, beginning at the occluso-buccal portions of the first molars, and having its highest point at the necks of the central incisors.

In the light of present knowledge, odd and isolated situations of areas of erosion can only be referred to localized gland affections; the glands overlying the spots of erosion being alone affected.

MORBID ANATOMY.

It is evident that we have to do with a chemical solution of calcium salts as the only distinctive feature of this disorder; the progress of the affection is essentially different from that of dental caries, in which chemical solution is but one feature. It is difficult to reconcile two apparently contradictory conditions in connection with erosion. It is

believed that only an acid substance can cause the decalcification, the molecular destruction of the enamel and dentin, and yet it is recognized that the application of an acid produces roughening of the tooth-surfaces; in erosion the surfaces are frequently highly polished.

FIG. 220.

Cases of erosion of the lower anterior teeth (drawn from a cast prepared by E. D. Swain, of Chicago): *B*, silhouettes representing the loss of substance in five of the affected teeth: 1, right lateral incisor; 2, right central; 3, left central; 4, left lateral; 5, left cuspid. The lines *a*, *a*, *a*, *a*, *a* show the position of the margin of the gum. A line is drawn also to show the original form of the tooth. (Black.)

Miller[1] states that "eroded teeth preserve their polish upon calcining, which shows that the polish pertains to the inorganic constituents of dentin."

The general progress of the decalcification is as shown in Fig. 220. The enamel is first cupped out, usually in a transverse groove, which maintains its general form, increasing in depth and width until the dentin is exposed; if the carious process be not grafted upon erosion, the excavation does not materially alter in form. The dentin when exposed is usually firm and polished, and in many cases assumes a translucent appearance, indicative of structural changes.

FIG. 221.

General direction of striæ of Retzius.

The shape of the excavation in the enamel appears to bear a somewhat close relationship with the striæ of Retzius. These striæ (see Chapter VII.) are lines in the enamel which appear to represent an orderly, regular, and periodical deposit of pigment in the forming tissue, and their distribution is of such character as to mark the size of the enamel-cap at different periods of its formation (Fig. 221). The presence of the pigmentary material, whatever it may be, leads to the inference that these lines in the enamel differ in some degree in chemical composition from the other portions of the tissue; there is possibly, or probably, some degree of difference in the comparative solubilities of the pigmented and non-

[1] *Dental Cosmos*, 1894, p. 269.

pigmented enamel. Color is lent this view if a careful examination be made of the form of the excavation in the early stages of a typical case of erosion. The base of the eroded cavity appears to lie upon successive striæ of the enamel. The decalcification appears to proceed inward until one of these striæ is met, which, furnishing an increased chemical resistance to the action of the decalcifying agent, causes the process of decalcification to proceed more rapidly along the lines between the striæ. Williams[1] has shown that the decalcification of enamel in caries finds lines of greatest chemical resistance in lines of enamel-stratification. It is possible that some of these lines irregularly situated may govern the direction which the erosive process may take; but characteristically they appear first to be checked and directed by the striæ.

SYMPTOMS AND DIAGNOSIS OF EROSION.

So far as the enamel is concerned, erosion can, of course, present no symptoms; it can only exhibit signs, which are the excavations themselves, their forms, and the characters of their walls. When the dentin becomes exposed an annoying hypersensitivity is occasionally observed. An examination of the lip will usually reveal the labial follicles to be enlarged; and if the mucous membrane be dried, a strip of blue litmus paper laid over it, and the lip pressed, the contents of the follicles will be forced out. "If the test be made in the morning, before breakfast, the litmus will be reddened. This acid reaction is not marked during the day."[2]

The existence of erosion has become a valuable diagnostic sign for the general practitioner in his search for the nature of masked maladies from which patients frequently suffer. Obscure gout has been pointed out through dental indications alone, where the practitioner had before been baffled in his diagnosis.

TREATMENT OF EROSION.

The treatment of erosion divides itself under two heads: prophylactic and restorative; the prophylactic is again divided into local and general treatment. The problem of eradicating the cause of the disorder lies in a correction of the morbid glandular secretion. It is evident that if the irritation and altered secretion of these glands be due to some systemic cause, a disease of suboxidation, notably an affection of the gout order, a cure of the local disturbance involves the cure of the underlying systemic cause.

The effect of an anti-gout regimen and anti-gout therapeusis upon the advance of the erosion has not been sufficiently tested or observed to furnish reliable data in this connection; but so far as tests have gone

[1] *Dental Cosmos*, 1897. [2] Truman.

such treatment appears to lessen the formation of acid substances by the labial glands. Brubaker (*supra*) has suggested the advisability of destroying these glands by means of the electrocautery, as a radical cure of the progress of erosion.

Next in importance to the prevention of acid formation, is its neutralization. This implies the application of alkalies or the use of alkaline-mouth-washes. The greatest production of acid occurring during the night, applications of adhesive masses of alkaline substances are made to the teeth at night. The principal of these is prepared chalk, calcium carbonate; it is rubbed over the labial faces of the teeth and between them, before retiring. It remains in sufficient amount to neutralize any acid substances coming in contact with it.

Excellent results as to the checking of the progress of the decalcification are obtained from the use of magnesium hydrate[1]—precipitated magnesium hydrate held in suspension in water, milk of magnesia. Kirk found that three hours after the use of a teaspoonful of the milk of magnesia that the saliva maintained an alkaline reaction. It is deposited in a film over the surfaces of the teeth, making an alkaline coating. It should be used freely before retiring at night. If the patient care to take sufficient trouble, it is an excellent practice to dry the labial faces of the teeth each evening and paint them with a solution of amber in chloroform.

Restorative Treatment.—If the cavities of erosion are treated as cavities of decay, excavated and filled, it is found that the erosive process goes on around the edges of the fillings, leaving the latter projecting as metallic islands from the surfaces of the teeth. Many operators, deeming the insertion of gold fillings unwarrantable under the conditions, advocate and insert zinc-phospate fillings, replacing them as often as necessary. The filling-material itself undergoes a decomposition similar to that of the enamel, so that it becomes cupped out. Such fillings should be well polished and varnished. If the cavities are of proper form, porcelain or glass inlays[2] may be used with advantage. Like other fillings, they must be renewed when necessary. About the necks of the lower teeth the restorations are usually made with amalgam.

Enamel-stains.

Stains of the enamel are not to be confounded with deposits of deeply colored calculi, although there is no doubt some similarity in the chemical conditions which bring about the discoloration in both instances. The nature and differences between these pigmentary deposits were first set forth by Miller,[3] but the questions involved are still *sub judice*.

[1] Kirk, *Dental Cosmos*, 1893. [2] See *American Text-book of Operative Dentistry*.

[3] *Dental Cosmos*, 1894.

METALLIC STAINS.

Copper.—Miller found that "workers in copper, brass, or bronze all presented a green stain upon the upper teeth, showing every shade of green and bluish-green up to bluish-purple. The latter color predominated in rooms where phosphor-bronze was worked." Attention is called to the fact that "trumpeters very often show a discoloration of the teeth." Similar discolorations are sometimes noted in proximity to copper-amalgam fillings. The presence of copper was demonstrated in scrapings from some of the stained teeth, imparting a characteristic green color to a Bunsen flame.

Iron.—"Workers in iron presented stains of a brownish color." As pointed out, "the green salts of iron under the conditions found in the mouth would become oxidized and brownish in color." The administration of iron salts medicinally is believed to produce black discolorations, iron sulfid being formed. "Iron deposits are usual in the border-line between carious and normal dentin." It is usually believed that the brownish spots frequently seen in connection with incipient or arrested caries of the underlying enamel are due to the formation of iron salts.

Manganese.—Manganese was found in the dark-colored deposits upon the teeth of herbivorous animals, but as yet not upon those of man. The investigators state "that alkaline saliva may be necessary to the production of these deposits."

Mercury.—In cases of prolonged mercurial administration the deposits (black) upon the teeth may give the reaction for mercury. "If mercury and potassium iodid are given together, the green iodid of mercury might be present upon the teeth." It is probable in these cases that another discoloring substance may form. There is in mercurialism more or less gingivitis; the gums are swollen and spongy, bleeding readily. "More or less putrefactive decomposition of the albuminous matter present upon the teeth occurs, and hydrogen sulfid is formed. Reacting upon the oxyhæmoglobin of the blood, sulfo-methæmoglobin is formed—greenish-red in concentrated, green in dilute solutions." Miller ascribes the discoloration found in conditions of gingivitis from various causes, with lack of hygienic care, to a probable reaction between hydrogen sulfid and oxyhæmoglobin.

Lead.—Hirt (quoted by Miller) found in cases of lead-poisoning discolorations upon the teeth: dark brown at the necks, light brown on the crowns, with sometimes a trace of yellowish-green. Miller's tests (limited in number) showed no lead reaction from the dental deposits in lead-poisoning.

Nickel.—Some of the salts of nickel are green. "Metallic nickel attacked by fluids of the mouth and mixtures of bread and saliva pro-

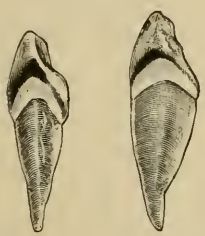
duce greenish salts. The writer has seen the entire root of a tooth [1] containing a nickel retaining-screw stained a uniform apple-green."

Silver.—The dentin of pulpless teeth containing amalgam fillings is sometimes stained black, owing to the formation of silver sulfid, but as yet no silver deposits upon enamel have been detected.

GREEN STAIN.

This most common of green deposits upon enamel occurs upon both the temporary and the permanent teeth of young persons. The deposits usually have a crescentic form, are mainly upon the labial faces of the anterior teeth, and may be but a narrow line or may cover one-half the labial face. It is unusual for the deposits to extend far into the interproximal spaces, their tendency being to follow the edges of the proximal surfaces. While green stain undoubtedly does form upon adult teeth (Figs. 222 and 223) where clearly the enamel-cuticle has long been

Fig. 222.



Extension of green stain on the approximal surface of the incisors. (Miller.)

Fig. 223.

Extension of green stain on the lingual surface of incisors. (Miller.)

absent, it is only very common upon young teeth where remnants of Nasmyth's membrane persist about their necks. The color of these deposits varies from light green to greenish-black.

If an instrument be passed over the portion of enamel affected, more or less roughness of the surface is evident. If the deposits are subjected to friction with abrasives, they disappear slowly and the enamel beneath is found roughened. This has led to the belief that these deposits cause decalcification of the enamel. It is found upon adult teeth that when an area of cervico-labial enamel has become roughened through slight decalcification, that green stain is likely to form upon the rough surface, if proper hygienic care be not exercised. It is also found that if the stain be removed by means of abrasives, the roughened enamel may be readily polished—*i. e.*, the decalcification is very superficial.

If cases be observed early enough in childhood, it will be noted that green stain is usually preceded by a lack of oral hygiene; collections

[1] Specimen in possession of E. C. Kirk.

of food-débris are not removed from about the necks of the teeth, which implies that prior to the formation of green stain the affected enamel-surfaces have been subjected to the action of fermenting food-débris—that is, to acids. These facts have led to a general acceptance of the view that the roughness or decalcification has preceded the green deposits. "If teeth be placed in a 10 per cent. solution of hydrochloric acid, in from two to four minutes the enamel-cuticle begins to loosen, and in from five to ten minutes is isolated. It is found that the entire stain comes away with the cuticle."[1]

Nature of Coloring-matter.—The coloring-matter is found to be insoluble in water, glycerin, alcohol, ether, chloroform, and oil of turpentine. Mineral acids, hydrochloric, nitric and nitro-hydrochloric, act but slowly upon the coloring-matter; even hydrochloric acid requires some hours to completely destroy it. Tincture of iodin, commonly believed to act as a solvent of green stain, was found to affect it but slightly. Both chlorin and nascent oxygen destroy the coloring-matter rapidly, the cuticles being bleached in a few minutes by a 10 per cent. solution of hydrogen dioxid. Thick, dark-green deposits were incompletely bleached after eight hours' immersion in the 10 per cent. H_2O_2 solution, pointing to a lack of uniformity in the composition of the stain.

The belief that the green coloring-matter is chlorophyll is contradicted by the fact that it is not soluble in ether.

So far as present evidence is possessed, the association of the green discoloration with sulfo-methæmoglobin, or some allied substance, is the most probable explanation.

Furthermore, there is no evidence that green deposits stand in causative relation to enamel decalcification; as pointed out, they are more probably deposits, following upon limited decalcification.

Treatment of Enamel-stains.—The general modes of treating staining of the dentin[2] are not of general application in the treatment of enamel-stains. The problem in the removal of metallic stains is to transform an insoluble metallic salt into a soluble one. The most frequent and most practicable course in dealing with metallic stains of the dentin is to form soluble chlorids through the action of nascent chlorin. Copper, nickel, and iron stains should be treated by repeated and continued washings with chlorin-water; silver the same, or be acted upon by iodin tincture, forming soluble iodid, and so on; but as all of these deposits, including green stain, are very superficial, the rational course of treatment is their mechanical removal by means of abrasives. A mixture of powdered pumice-stone and glycerin is used in conjunction

[1] Miller, *Ibid.*

[2] *American Text-book of Operative Dentistry.*

with rapidly revolving leather wheels, brushes, and rubber cups, until every vestige of the deposit has disappeared and the enamel surface is polished.

Enamel Solution.

Usually the first stage of dental caries consists of a solution of the calcium basis of the enamel; the decalcification is a distinctive phase of dental caries, and will be discussed under that head.

CHAPTER XII.

DISEASES OF THE DENTIN.

The dentin, as shown in Chapters VII. and VIII., is made up of a matrix of calcified tissue traversed by innumerable filaments or processes, which are vital protoplasmic prolongations of the peripheral cells of the pulp. The matrix of the dentin contains two distinct substances, one forming a thick and uniform coating about each tubule, so that the dentin is traversed by as many of the coatings or sheaths of Neumann as there are odontoblastic processes. In addition, the enclosing sheaths are united laterally with one another by a material akin to or identical with that forming the sheaths of Neumann. It is believed that the substance of these sheaths is a transitional product which, when fully calcified, becomes the basis-substance of dentin. Subjected to the action of acids, the material of Neumann's sheaths is found much more resistant than the basis-substance of dentin.

The relation of dentin to the odontoblasts is that of a formed product. So far as disease-processes are concerned, the formed material of the dentin must play a passive part; although this may and probably does not apply to the transitional substance, the sheaths of Neumann, as being but a partially formed tissue it is susceptible of constructive change. Disorders which involve constructive nutritive changes in the dentin can evidently be only associated with its vital parts, which are the dentinal filaments, prolongations of the peripheral cells of the pulp. It follows, therefore, that all nutritional changes which occur in the dentin are directly the result of a physiological or pathological process in the pulp; and associated disease-conditions are not diseases of dentin, strictly speaking, but are diseases of the pulp.

Classification.—What are termed diseases of the dentin, meaning diseases in which the structure of the dentin undergoes changes, are divisible into two classes—constructive diseases and destructive diseases. The constructive diseases in an accurate classification can only be those conditions in which formative structural changes occur about the dentinal tubuli. As transitional material exists, no doubt, also about the transverse as well as the longitudinal processes, and as partially calcified tissue probably occupies many of the interglobular spaces, at least the possibility must be assumed of a change which could affect all of this

material—*i. e.*, transform it from a partially to a completely calcified material.

The formation of adventitious deposits of dentin or modified dentin on the surface or in the substance of the dental pulp is clearly a process to be included under the diseases of the pulp proper.

The destructive diseases of the dentin are divisible, according to their causation, into, first, those due primarily to chemical action—erosion and caries; secondly, those due to physical forces—abrasion; thirdly, those due to vital causes—a resorption. In all of these the basis-substance of the dentin must be regarded as playing a passive part—it is a formed material being acted upon.

Abrasion of the Dentin.

The nature and character of abrasion of the teeth have been discussed under the head of affections of the enamel. Its progress is more rapid than is that of enamel abrasion. It is usually attended, particularly in cases of abrasion about the necks of the teeth, by an increase in the sensitivity of the dentinal fibrillæ, and pulp reaction.

Erosion of Dentin.

The solution or destruction of dentin in the progress of erosion appears to be much slower than its progress in enamel; as in abrasion, a heightened sensitivity of the dentinal fibrillæ is common.

Secondary Deposits.

It is noted in both abrasion and erosion, particularly if the progress of the affections be slow, that the dentin undergoes changes in appearance which are quite characteristic; instead of having the semi-opaqueness of the dentin, this tissue acquires a horn-like translucency. When the teeth are broken open it is common to find the pulp has receded from its position opposite the abrasion or erosion, the area of withdrawal being occupied by a deposit of dentinal substance upon the wall of the pulp-chamber. In cases of coronal abrasion the deposit may be uniform, reducing the size of the pulp-chamber at all aspects.

It is the change from opacity to translucency which is the notable feature. This change is also noted normally in the dentin of aged persons. It has been emphasized by Miller[1] that the opacity of the dentin is due to the differences between the light refractive index of the basis-substance of the dentin and that of the dentinal tubuli. Were both of these of uniform composition, the dentin would be translucent. It is a rational inference, therefore, that the nearer to a uniform composition the dentin attains the more translucent it becomes. Now, as it is

[1] *Micro-organisms of the Human Mouth.*

physiologically out of the question that the basis-substance or formed dentin should become identical with the contents of the dentinal tubuli, there is but one inference, which is, that the tubuli acquire the nature of the formed dentin. It is stated, in objection to this opinion, that if the dentin be acted upon by dilute acids, it becomes translucent. In this immediate connection Miller[1] noted that the zone of translucency (see Chapter XIV.) in dental caries was not caused by decalcification, as that portion of the dentin contained an excess of calcium salts. These facts are in themselves sufficient to indicate that the appearance of translucency in dentin is due to changes which obliterate the dentinal tubuli; inferentially which cause calcification in the sheaths of Neumann and a recession of the odontoblastic processes. As a matter of fact, the dentinal tubuli certainly do normally decrease in calibre with age.

Positive data are wanting as to the direct causes of the appearance of translucency and as to exactly how the translucent dentin differs from ordinary dentin in anatomical and chemical composition. From the examples cited, however, the evidence points to a constructive reaction of the protoplasmic portions of the dentin, to a continued stimulus; it is a physiological defence of the pulp. It is an expression of cell-stimulation which evinces itself in a heightened functional activity—*i. e.*, increased dentin-formation; the hypersensitivity of the dentin is another evidence of increased functional activity. A similar or identical process is noted in connection with what is called the spontaneous arrest of dental caries (see Chapter XIV.).

Resorption of Dentin.

Some of the features of this process will be discussed under the head of diseases of the pericementum, "resorption of the roots of permanent teeth." The normal resorption of the roots of the temporary teeth and the resorptions which occur upon the root-surfaces of implanted, replanted, or transplanted teeth, are a species of phagocytosis; the tooth-tissue being removed piecemeal through the action of cells—odontoclasts—which are in all probability modified leucocytes. A similar process is sometimes noted when the tissues about the gum-margin and the marginal pericementum are in a state of localized irritation or inflammation.[2] In interdental spaces where large fragments of wooden toothpicks have been broken off, there has been observed a deep erosion in the cementum and in the dentin of the tooth, the enamel apparently being unaffected. The appearances in no wise resemble those of caries. In one case such an accident as cited resulted in resorption of the lateral neck-walls of an upper cuspid and bicuspid, exposing the pulp-chamber in a few weeks.

[1] *Micro-organisms of the Human Mouth.* [2] *American System of Dentistry*, vol. i.

Black[1] speaks of the process as resulting from mild irritation of the soft tissues, stating that as soon as pus has formed the absorptive process ceases. These absorptions have been noted when the evidences of traumatic injury were marked, there being, however, no evidences of pus-formation. These were, in all probability, cases of phagocytic reaction in which the dental tissues were secondarily involved. The exact nature of the solvent formed by the cell, which brings about the solution of hard tissues, is not known. Krause has suggested that it may be lactic acid. So far as the nature of the process can be made out, it is due to the action of multinucleated phagocytes, which attach themselves to any substance to be removed, bone, dentin, etc., and resorption follows.

Resorption of Pulp-chamber Walls.

Gaskell[2] has reported a case where a central incisor entirely free from caries exhibited on its palatal aspect a pinkish tinge, which increased in depth until the enamel overlying crushed in, revealing the pulp of the tooth lying immediately beneath; there had been a resorption of a large mass of the dentin lying between the pulp and the enamel. The pulp was removed and the tooth filled. No history is given as to the condition of the root, whether resorption had occurred there or not. Shortly after, the adjoining central incisor exhibited a like pink coloration, which increased, leading to the inference that resorption was in progress in this tooth also. At the suggestion of E. C. Kirk the patient received continued doses of arsenic iodid and the compound syrup of the hypophosphites, in the hope of inducing a general and local constructive metamorphosis. This treatment was followed by a gradual disappearance of the pink coloration, an evidence of a redeposition of dentin. In the absence of histological data it is impossible to state just what was the nature of the repair-tissue in this case.

[1] *American System of Dentistry*, vol. i.

[2] *Proc. Academy of Stomatology*, Phila., 1895.

CHAPTER XII. (Continued).

DENTAL CARIES.

The last member of the group of destructive diseases of dentin is the all-important one of dental caries, the most universal of all diseases, and one from which but few civilized persons are entirely exempt.

Definition.—Dental caries may be defined as a progressive molecular destruction of the calcic tissues of the teeth, the first stage of which is a solution of the calcium salts by lactic acid ; the second, the dissolution of the organic matrix through the agency of saprophytic fungi. Definitions have varied according to the contemporary knowledge of the etiology and pathology of the disease ; the above is a pathological definition.

History.—Examinations of crania show the disease to be certainly as old as semi-civilization, and when more data are obtainable it will, no doubt, be found even older. The skull of a mummy in the British Museum, dating 2800 B. C., exhibits well-marked caries and other dental diseases. Caries appears in the teeth of the skulls of all peoples, no matter what their degree of civilization, provided their dietary included cooked starchy foods.

The explanations as to the nature of dental caries have undergone changes parallel with the advances of the collateral sciences of surgery. In each age or generation theories as to the origin and natural history of dental caries have reflected the contemporary state and condition of the science of pathology ; with a modification of the theories of pathology have come changes of conception as to the essential nature of the dental disorder. At the present time the etiology—at least the direct etiology—of dental caries constitutes one of the best examples in all medicine, of absolutely demonstrable scientific facts.

In its indirect etiology, and in some features of its pathology and morbid anatomy, there is much left to be discovered ; although Miller has settled beyond doubt the question of its direct causation. Miller's discoveries and demonstrations were, as all great discoveries in any field of science, the outcome, or an evolution from all that had gone before. Theories were partially formulated and set forth by previous investigators, but it was he who furnished what science rigorously exacts from all her workers—direct and complete demonstration substantiating a formulated theory.

Investigations and opinions prior to the writings of John Hunter, interesting though many of them be, are necessarily purely speculative

in nature, for the reason that before Hunter pathology bore but a faint resemblance to a science, as science is now understood. Hunter,[1] while apparently inclined to classify dental caries with inflammatory disturbances, had sufficient doubt of such a position to note that caries began upon the outsides of teeth. The next English writers upon odontography, Fox and Bell, both regarded dental caries as an inflammatory disturbance, Fox believing that the disease attacked first the "lining membrane" of the pulp-chamber, and that the dentin deprived of its source of nutrition died and disintegrated. Bell[2] vigorously combated the theories of his predecessors, and set forth positively that caries always began upon the periphery of the dentin, maintaining that the white and colored spots upon enamel arose secondarily, and were evidences of underlying carious dentin. Also, that caries was dental gangrene, and proceeded from the periphery toward the pulp. He believed that as soon as the pulp and crown were destroyed the caries ceased, and that the roots became of the nature of foreign bodies and were cast off.

Until 1830 nearly all writers subscribed to a greater or less extent to the inflammatory theory of caries, which received its final blow in the observation that the natural crowns of teeth which had been mounted on plates decayed. The only vestige remaining at the present of a belief in the inflammatory theories of dental caries is the writings of Bödecker and Abbott, although their opinions have a broader foundation than those of the older observers mentioned.

These theories may all be set aside, since it is recognized that dental caries proceeds in teeth whose dentin has been deprived of its source of nutrition. It occurs in pulpless teeth; in human and other teeth mounted upon plates; and, moreover, may be artificially produced outside of the mouth.

The next theory was radically different from the foregoing. In 1835 Robertson, of Birmingham, England, advanced the opinion, based upon his observations, that it "is to chemical and not to inflammatory action that the destruction of the teeth must be attributed." The author points out forcibly the errors and fallacies of previous writers. He states that[3] "Particles of food retained in fissures and imperfections of the teeth and in the spaces between the teeth undergo a process of decomposition and acquire the property of corroding, disuniting, and therefore destroying the earthy and animal substances of which the teeth are composed."

John Tomes, a little later, was the first to record microscopic examinations of carious dentin. He described the transparent zone lying

[1] *Practical Treatise on Diseases of the Teeth*, 1778.

[2] Bell on *The Teeth*, Phila., 1830.

[3] Robertson, *A Practical Treatise on the Human Teeth*, 2d ed., Phila., 1839.

between the carious and non-carious dentin, and observed and pointed out also the dentinal fibrillæ. He announced the very significant fact in relation to caries, that[1] if blue litmus paper be applied to a carious cavity it is at once reddened, which furnishes evidence of the presence of an agent capable, if unresisted by the vitality of the dentin, of depriving the tissue of its earthy constituents, leaving the "gelatin to undergo a gradual decomposition favored by the heat and moisture of the mouth."

Tomes first established the essentially chemical character of some features of caries. The character of the acid and its localization were, however, not ascertained.

The electro-chemical theory of Bridgeman was the next hypothesis advanced; from observation he noted that caries was more frequent in moist than in dry mouths, and that caries occurred more readily about some types of fillings than others. He believed that the tissues of the gum acted as the negative pole of a battery, the enamel of the tooth-crown as the positive element, and the fluid in which they were bathed as an electrolytic fluid. The difference in the electric potentiality of soft tissues and the tissues of the tooth-crown was deemed sufficient to supply the conditions of a battery. Acid substances being set free at the positive pole, acted as decalcifying agents. The source of the acid was not held to be from the electrolytic fluid, but formed by acid radicals set free from their combination with the calcium salts of the enamel. It will be seen that this theory is substantially the basis of the compatibility theory of recurring caries.

It has been maintained, principally by S. B. Palmer, that the cause of the recurrence of dental caries about dental fillings is due to the establishment of artificial electro-chemical relations; the readiness with which the tooth-walls succumbed being governed by the extent of difference of electric potentiality between filling-material and dentin. Recurrence of decay appeared to occur most readily about gold fillings, less so about tin, and least about gutta-percha, the explanation given being that the difference of electric potentiality is greatest between gold and dentin, and least between dentin and gutta-percha. The fluids of dentin, called of poor structure, were regarded as electrolytic in a battery of which one element was the filling and the other the dentin; the moisture of the dentin being electrically decomposed, oxygen (and acids) were set free, which acted as the destructive agents. These opinions, set forth by Palmer in 1874, were reiterated by him in 1894.[2]

Miller[3] examined these assumptions, and reported a long series of experiments relative thereto. He states that as dentin is a non-conductor it cannot form an element of a battery; the fluid with

[1] *Dental Surgery*, 1859. [2] *Dental Cosmos*, Nov., 1894. [3] *Ibid.*, 1881.

which dentin is permeated is, however, a conductor. "It is always possible to produce an electric current by means of two liquids and one metal, provided that both liquids are conductors, communicate freely with one another, and act differently upon the metal. Hence, an amalgam or tin filling in a tooth with the saliva upon one side and the fluids of the dentin upon the other would produce a stronger current than with gold." "Living dentin is a better conductor than dead; hence in such case metal fillings should fail sooner in contact with vital than with dead dentin." "Assuming that for the production of an electric element it is only necessary that one of them be more easily acted upon chemically than the other; enamel and dentin, dentin and cementum, are such substances; hence a tooth is a galvanic battery when in contact with saliva."

Pieces of ivory and dentin, having the different filling materials inserted in them, were suspended in dilute acid for periods of weeks, and at the end of that time all were found affected by the acid in about equal degree; had electrolytic currents been generated between the metals and dentin the latter would have been acted upon more vigorously in some cases than in others.

Prior to this time the chemical theory of dental caries had received general acceptance, the question of the origin of the acids being still in doubt. Before 1868[1] George Watt had advanced the mineral-acid theory: that decay is caused by the action of mineral acids (nitric, hydrochloric, and sulfuric), generated in the mouth, upon the calcic tissues of the teeth. Watt, using the older chemical nomenclature, ascribes the origin of sulfuric acid to be from H_2S generated in putrefactive processes; it is acted upon by oxygen; sulfur is set free, which in a nascent state combines with oxygen, forming sulfur dioxid, in the presence of the watery saliva becoming SO_3 (old nomenclature), sulfuric acid.

"Hydrochloric acid may be free in the mouth, or may result from the decomposition of chlorids. Chlorin is set free, which, combining with hydrogen, forms HCl."[2]

Nitric acid was held to be formed from the ammonia produced in the process of organic decomposition; decomposed by the action of oxygen, nitrogen oxids are formed, one of them being nitric acid.

The physical appearance of the carious dentin was held to be due to the offending acid; if "sulfuric acid, black decay was produced; if nitric, white decay; hydrochloric acid producing the yellow and brown decay."[3]

It is natural that in the country of Pasteur the organic acids and the sources of their origin in fermentation should be advanced as the possible causes of tooth-decalcification. Magitot pointed out that the

[1] *Chemical Essays*, 1868. [2] *Ibid.* [3] *Ibid.*

essential phenomena of caries, as they were then understood, were the same in natural teeth mounted upon plates as in the natural organs *in situ;* proving that caries is intrinsically independent of the existence of vitality. By immersing teeth in solutions of sugar undergoing fermentative changes he found that decalcification occurred. Teeth immersed in solutions of sugar in which fermentation had been prevented by boiling the solution and sealing, or by additions of sufficient carbolic acid, remained unaffected.

Leber and Rottenstein, in 1867, first called attention to the probable causative association of bacteria with some phases of dental caries. By staining carious dentin with iodin the dilated dentinal tubules were shown to be filled with granular bodies, which they recognized as bacteria, identifying but one of the many forms of oral bacteria—the leptothrix. They deemed an initial exposure of dentin a necessary preliminary to the invasion and growth of the leptothrix, which in conditions of lessened resistance gained access to the tubules and in some undescribed manner caused their dilatation.

The question of the recognition of the presence of bacteria directly resolves itself into the subject of special staining. Prior to the work of Koch, presented in 1881, no means of isolating specific bacteria by special cultures and staining were known, and it is remarkable that in the same year the essential features of dental caries were first made out with some degree of clearness.

Milles and Underwood (World's Medical Congress, 1881) point out clearly and at length the different appearances produced by simple decalcification of dentin and those by dental caries. Speaking of Magitot's experiments, they say: "We assume that two factors have always been in operation: (1) the action of acids, and (2) the action of germs. When caries occurs in mouths it is always under circumstances more favorable to the action of germs than to the action of acids." They believed that the acids necessary for the decalcification were excreted by the germs, which utilized the dentinal fibrillæ as a food-supply.

It will be seen that the invasion and multiplication of organisms in the tubuli were held as the antecedent of the process of decalcification. The deductions of these gentlemen were drawn from data not derived from the methods of modern bacteriology—*i. e.*, special stains and special cultures. Moreover, they were made before the physiological chemistry of bacteria was even partially understood.

In 1882 W. D. Miller, of Berlin, announced as the results of experiments conducted by him that he believed the first stage of dental caries to consist of a decalcification of the tissues of the teeth by acids which are for the greater part generated in the mouth by fermentation. This, it will be seen, is a position in agreement with that of Leber and Rotten-

stein, rather than with that of Milles and Underwood. Miller's experiments[1] carry conviction with them.

Experiment 1.—Fresh saliva, mixed with starch (1 : 40) and kept at blood-temperature, invariably became acid in from four to five hours.

Experiment 2.—A glass tube, 2 cm. long and 3 mm. wide, was filled with starch and fastened to a molar tooth on going to bed; the next morning the contents of the tube had a strong acid reaction.

Experiment 3.—When mixture was heated for half an hour at 100° C. and placed in an incubator it did not become acid in twenty-four hours.

Experiment 4.—When the starch alone was heated to 150° before mixing, the solution became sour; hence the ferment exists in the saliva, and not in the starch.

Experiment 6.—Carbolic acid was added to saliva and starch mixture to $\frac{1}{2}$ per cent. strength, and put in an incubator: when tested in a few hours no acid was found, but sugar was in solution.

Experiment 7.—A mixture of saliva and grape-sugar was subjected to a temperature of 67° C. for twenty minutes (which destroys ptyalin, but cannot destroy organized ferments), and placed in an incubator for twenty hours: the solution became acid, hence the fermentation was caused by an organized ferment.

Experiment 8.—Several drops of a solution of starch and saliva (1 : 40) were put in each of several sterilized test-tubes and sterilized. One tube was used as a control. One tube was infected with carious dentin: in twenty hours the solution was acid. From this tube a second tube was infected; from the second a third, and so on: each became acid—*i. e.*, an organized and reproductive ferment, producing acid, was contained in deep layers of carious dentin.

Other experiments demonstrated the fungus to be independent of the free access of oxygen for its development. Infections by saliva of the above named mixture made before and after a vigorous cleansing of the teeth showed that the amount of acid produced after cleansing was often not more than one-fourth that produced by the saliva before cleansing the teeth.

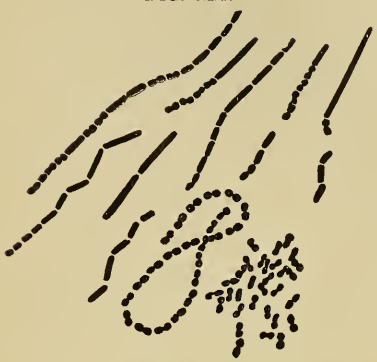
Cultures from the deeply infected dentin showed the growth of organisms (Fig. 224) identical in form with the bacillus acidi lactici. Grown upon carbohydrates, acid solutions were produced; grown upon beef-extracts without carbohydrates, no acid reaction took place.

Sections of sound dentin were placed in 5 c.c. of a neutralized 2 per cent. solution of beef-extract. In a second test-tube similar sections were placed in the same solution with the addition of 0.2 per cent. of cane-sugar; both were sterilized, and then infected with a pure culture of the fungus under discussion. After weeks the first tube gave no acid

[1] *Independent Practitioner*, 1884–'85.

reaction, and the dentin sections gave no evidence of softening. In the second tube, that containing the cane-sugar, all of the sections were entirely softened within three weeks, and stained sections, mounted, showed the tubes to be invaded by organisms and dilated, in some places the walls being broken down, forming caverns.

Fig. 224.



Fungi from carious dentin. (Miller.)

By inducing and checking fermentation after a mixture of saliva and starch had acquired an acid reaction, filtering, and accumulating the filtered acid solution until a litre had been obtained, Miller sought to determine the nature of the acid present. Its volume was reduced over a water-bath to 75 c.c.; a few drops placed in a dilute solution of methyl-violet produced no change—*i. e.*, the acid is organic. Concentrated over the bath to 40 c.c., the solution was next shaken with one and one-half to two litres of ether, and allowed to stand until the ether became transparent; this was then distilled until the volume was reduced to 50 c.c. The filtered solution was further reduced over the water-bath. An excess of freshly prepared zinc oxid was added, the solution boiled, and water added until the reaction became neutral. Filtered again, the solution was set aside until crystallization occurred; a drop placed upon a slide under the microscope showed the forms (Fig. 225) of crystals of zinc lactate. By testing the molecular weight of the washed and dried crystals it was determined clearly that the substance was zinc lactate.

Fig. 225.

Miller obtained lactic acid from carious dentin directly. The carious dentin of several freshly extracted teeth was freed from food-debris, cut in fine pieces, placed in a test-tube with 1 c.c. of water, and 2 drops of a 10 per cent. solution of hydrochloric acid added. Twenty-five c.c. of ether were then added and after some minutes the ether holding the lactic acid present in solution was poured off and left standing in a test-tube twenty-four to forty-eight hours, until the solution was clear. Filtered and evaporated, a few drops of distilled water and zinc oxid were added: crystals recognizable as those of zinc lactate formed.

The character of the infecting organisms is shown in the following

manner "a beef-sugar extract is infected from the deep layers of carious dentin; the solution is kept free from extraneous organisms and at a temperature of 37° C. The solution clouds in a few hours; in fifteen hours the fermentation will have reached its most active state; the solution soon after begins to clear, and a flocculent colorless precipitate collects as a sediment upon the bottom of the vessel. The sediment consists of cocci and micrococci, single or in chains (Fig. 226)." These

FIG. 226.

fungi cause the direct splitting up of sugar without the formation of carbon dioxid, $C_6H_{12}O_6$, a molecule of glucose, forming $2C_3H_6O_3$, or two molecules of lactic acid.

The fungi have the power of inducing hydration of non-fermentable cane-sugar, converting it into fermentable levulose and dextrose—

$$\underset{\text{Cane-sugar.}}{C_{12}H_{22}O_{11}} + H_2O = \underset{\text{Levulose.}}{C_6H_{12}O_6} + \underset{\text{Dextrose.}}{C_6H_{12}O_6}$$

Miller at this time deduced from his studies that the nature of the carious process is as follows: "Whenever solutions of sugar (nearly always present in the human mouth) stagnate in fissures between the teeth, etc., they *must* become acid. The acids gaining access to the dentin decalcify a portion of that tissue; the tubules of the decalcified dentin take up the solutions of sugar and organisms which develop independent of the access of air. Flourishing they produce lactic acid within the tubules. As each layer of dentin becomes softened in time, the micro-organisms *follow after*, continually producing new acid."

The same observer has added much to this basal doctrine since his original publication, which matter is considered under appropriate heads. The discoveries set forth are the demonstrations which convinced the dental fraternity that the essential character of the carious process had at last been made out. There are matters relative to the

etiology, pathology, and clinical history of caries which still require elucidation.

Williams[1] has supplied a missing link in the pathology and morbid anatomy of caries by demonstrating the details of caries of enamel. Black[2] has exploded several fallacious ideas previously held as to the etiology and clinical history of caries, and paved the way for a better understanding of the predisposing causes and variations of the active causes of dental caries.

Some of the features of the morbid anatomy of dental caries, as shown by Miller, have been expounded by Heitzmann, Bödecker, and Abbott, who represent the contemporary school of believers in the inflammatory origin of dental caries. The substance of the opinions of these gentlemen is that dental caries consists in a return of the dentin to its embryonic condition. The observation that dental caries may be artificially produced and occurs in dead dentin, exhibiting the same features as ordinary carious dentin, is, of course, the death-blow to this inflammatory theory.

[1] *Dental Cosmos*, 1897. [2] *Ibid.*, 1895.

CHAPTER XIII.

DENTAL CARIES: ITS CAUSES AND CLINICAL HISTORY.

The causes of dental caries are both predisposing and exciting. Each of these again may be subdivided into general and local.

Exciting Causes.

Although it is usual to discuss the predisposing causes of a particular disease before passing to a description of its exciting causes, the predisposing causes of dental caries are made more clear when the nature of its exciting causes is first understood.

It has been demonstrated (see Chapter XII.) beyond reasonable doubt that the direct exciting cause of dental caries is lactic acid. A discussion of the exciting causes, therefore, involves the full consideration of the conditions of lactic fermentation in the human mouth. This includes two factors: first, the substances out of which lactic acid is formed; and, second, the organisms which cause the transformation.

Lactic acid is formed out of—*i. e.*, requires for its production the presence of—substances identical with, or which are under the conditions of the mouth converted into, glucose, $C_6H_{12}O_6$. These are the three groups of carbohydrates—glucoses, $C_6H_{12}O_6$, saccharoses, $C_{12}H_{22}O_{11}$, and amyloses, $C_6H_{10}O_5$. Those most common in the human dietary are grape-sugar or dextrose, and fruit-sugar or levulose, in the glucose group; cane-sugar in the saccharose group; starch, cellulose, and gum in the amylose group.

Cane-sugar becomes converted by hydration into the two fermentable carbohydrates levulose and dextrose, both having the same composition, $C_6H_{12}O_6$. This hydration is brought about by the action of organized ferments, such as ptyalin and amylopsin, which convert starch into glucose,

$$\underset{\text{Starch}}{C_6H_{10}O_5} + H_2O = \underset{\text{Glucose.}}{C_6H_{12}O_6}.$$

In order to effect the localized and persistent fermentation of carbohydrates necessary for the production of dental caries, the question of lodgement of ferments and fermentable materials is all important.

Given points of lodgement (see Predisposing Causes of Caries) with the presence of fermentable material, the next factor is the existence and proliferation of the ferment. Soluble carbohydrates, as cane-sugar (saccharose), lactose, and glucose, soluble in saliva, have their solutions carried into all spaces in and about the teeth; and if ferments be present, organized ferments as bacteria, the solutions furnish a food-supply to these organisms. Insoluble or but partially soluble carbohydrates, on the other hand, alter the conditions. The effects of the general oral distribution of soluble carbohydrates may be seen in the mouths of confectioners, who work amid sugar, in which evidences of general fermentation—the widespread decalcification of enamel and dentin—are observed. The same effects are apparent in children who are allowed to use sugars unstintedly.

Perfectly soluble carbohydrates are, however, in the majority of cases washed away by currents of saliva, and are in all probability removed almost entirely from about the teeth under normal conditions. On the contrary, insoluble carbohydrates, as solid substances, are not washed away, except mechanically, unless they are transformed into soluble substances and are in situations where they can be subjected to the irrigating force of the saliva. It is with these substances that the process of caries is probably most directly associated.

Caries, as has been pointed out, is not entirely a disease of civilization; some savage and barbarous races are affected, and ancient races have also been affected. It is, however, largely a disease of civilization and semi-civilization; so that the causes of its prevalence are to be sought for in the artificial environment of civilization. A notable difference between civilized and uncivilized races is in food-habit; in this connection, the food-habit as regards carbohydrates is referred to. Judging from the data thus far presented by dental ethnologists, the barbarous and semi-civilized races which suffer from dental caries appear to be those whose dietary includes *cooked* starches.

Starches in a raw state, in uncooked vegetables, are combined with fibrous vegetable tissue, the starch being enclosed in capsules of cellulose. The starch is not in condition to agglutinate; its containing structures crushed by the teeth, set free some of the starch-particles, which are mechanically separated from one another; these particles, acted upon by the ptyalin of the saliva, are hydrated, becoming glucose. The masses are in large part or wholly washed away by the saliva and mechanically carried away by the vegetable cellulose of plant-skins, etc. By cooking, the cellulose-coverings of the starch-particles are burst; the starch-particles are set free and collect in glutinous masses, which when taken as food are in condition to collect and remain in spaces,

fissures, etc., where, being acted upon by ptyalin, glucose is formed, furnishing localized fermentable masses. There is another important consideration in this connection: many food-substances, notably fats, are taken at a temperature at which they are wholly or nearly fluid; when taken into the mouth the reduction of temperature and other conditions lead to their collection on, around, and between the teeth, which collections not only undergo changes themselves, but serve as a retaining medium for other débris.

The second factor in fermentation, the first being the fermentable material, is the ferment itself. The conditions of the human mouth, under normal circumstances containing food-débris, an abundance of water, dead tissue, and a constant temperature, are such as to permit the growth of numerous micro-organisms. Variations in the conditions present favor or deter the development of some forms. Until the physiology of all the oral bacteria is clearly made out, the nature of the conditions favorable or deterrent to the active growth of definite forms can be but partially understood. One essential to the growth of the lactic ferment or ferments is of tolerably constant presence in the mouth, and yet the extent, nature, and rapidity of progress of dental caries vary widely in different individuals and apparently in different teeth of the same individual, and at different times in the same person. This leads to the inference that there are conditions which favor the growth of lactic ferments, and others which check it. Noting the association of the rapid progress of dental caries with general disease-states, it is inferred that these latter establish oral conditions which modify the growth of the lactic ferment. Again, in the absence of evident general disturbances, it is believed that local oral conditions which favor growth are established and disappear.

Predisposing Causes.

Conditions are noted which appear to bear a constant relationship to the progress of dental caries; when these conditions exist caries is liable to occur, and when they are absent it is usually slight or absent. These conditions are both general and local. Many of them, as just pointed out, are veiled in obscurity, and we have at present no means or data for determining the scope of their action. For example, why a denture almost free from caries for thirty years or more should suddenly fall a victim to the disease, in the absence of any change in the food-habit or any local anatomical changes, and with no apparent alteration in general nutrition, is beyond our knowledge. It is rational, however, to infer that conditions are present which favor the unusual development of the lactic ferment. The problem is one which belongs in the category of pathological chemistry of the cells of the entire body.

GENERAL PREDISPOSING CAUSES.

There are certain conditions in which caries is almost certain to increase. The most constant of these is pregnancy. Not only does caries increase in extent during gestation, but the rapidity of its progress is markedly increased. In many of the cases there is a notable change in the dietary of the individual and commonly a lack of the usual hygienic care, which serve to explain the increase of caries. In the absence of these causes, it is evident that the explanation must be found in one of two conditions, or in a combination of them. Either attack must be increased, or resistance lessened, or both occur concurrently.

The question of resistance is the resistance of the tissues of the teeth. It has been stated and maintained that during gestation there is a lessening of the calcium salts of the teeth, the reason being that calcium salts are robbed from the tissues of the mother to supply the tissues of the fœtus, and that the dentin suffered as do the bones of the body. The enamel being a non-vital tissue, of course could not be affected, although even decalcification of this tissue has been held to occur as part of the inverted nutrition. The mechanism through which resorption occurs is not set forth by believers in this doctrine. This process might act in one of two ways; vital cells, of course, being a necessity for the hypothetical process. The odontoblasts upon their surfaces and in their filaments must be the medium through which the retrograde metamorphosis is accomplished, or else the usual resorptive cells must be the active agents—multinucleated odontoclasts. Now, as it is never maintained that the matrix itself loses its form, it is evident that the cells must then cause resorption through the dentin-substance. A solvent must be formed capable of abstracting the calcium salts from the dentin, and capable of acting through the thickness of the transformed matrix. The calcium salts taken up by the odontoblasts, or odontoclasts, must pass through them, to be taken up by the veins. It was also held that after the period of lactation a redeposition of calcium salts occurred which restored the original composition of the dentin The mode of formation of dentin and the character of its nutrition are in themselves sufficient to set aside any such hypothesis as the above. In addition, Black has shown [1] that there is no evidence to support a belief in the lessening of the amounts of calcium salts of the teeth during pregnancy, so that the matter of calcium resorption during pregnancy meets the fate of the inflammatory theory of caries. It may be mentioned, in addition, that pulpless teeth appear to break down rapidly in the caries of pregnancy, and as there could not by any

[1] *Dental Cosmos*, 1895.

possibility be a retrograde metamorphosis of dead dentin, the amount of calcium salts in the tissue could not be a factor.

The remaining factor for consideration is the condition of the vital parts of the dentin. To what extent the condition of the dentinal filament is an element modifying the progress of dental caries is not made out. Do variations in the physiological condition of these protoplasmic filaments influence and modify the progress of dental caries? In many or most of the cases of outbreaks of caries it is difficult or impossible to disassociate an increase in the local causes of caries. In pregnancy, in such debilitating diseases as typhoid fever and tuberculosis, and in other wasting disorders, there is unquestionably an increase of dental caries, with an increased rapidity of progress. It is this element of the comparative rapidity of the carious process which leads to the inference that the condition of the vital parts of the dentin is a governing or modifying factor. Caries not being solely a chemical process, the solution of the calcium salts being but one phase of it, and the disorganization of the dentin-matrix another, it remains to be determined what *rôle* the dentinal protoplasm plays. It succumbs during the progress of caries, and probably, like all protoplasm, offers its vital resistance to the action of irritants. Since it, as other protoplasm, is profoundly affected by the conditions of general nutrition, does it offer a lessened resistance to the carious process? Clinical records appear to indicate that it does. Being at the extreme peripheral nutritive zone, its power of resistance would be likely to be much less than that of more freely nourished parts, those having a vascular system.

Similar conditions appear to prevail in the anæmic and leukæmic states. It is notorious that caries runs a riotous course in diabetic patients (glycosuria); the formation of lactic acid in the mouths of these patients is abundant and widespread.

Among the important general predisposing causes of dental caries must be placed heredity. Unquestionably the children of parents whose teeth have succumbed readily to dental caries are prone to be affected in a similar manner. Moreover, in many of these cases local conditions (environment) are not sufficient to account for the lessened resistance; the evidence points to a general condition underlying the dental vulnerability.

LOCAL PREDISPOSING CAUSES.

What are termed the local predisposing causes of dental caries are much more evident than the general; they include, first, variations in the structure, arrangement, and forms of the teeth, together with other local anatomical and physiological variations; they are not actual causes,

but favoring conditions. The variations of structure are macroscopic and microscopic, and, no doubt, variations in the chemical organization of the dental tissues also have a part. Any variation of structure which furnishes a space in which fermentable material can find lodgement is included under this head. The first of these includes fissures of the enamel; these are lines of faulty enamel-formation, marking the bases of the cusp-segments of the teeth. Their capability of acting as elements of predisposition is in proportion to their depth and extent. They are most marked, as a rule, in the lower molars, next in the upper molars and bicuspids; next in the lower bicuspids. Pits are another favorable lodgement-place for débris; these are found most frequently at the bases of the enamel-girders of the upper incisors, at the extremities of the sulci of bicuspids, and at the bases of the anterior cusps of the upper molars, and marking the extremity of the buccal sulci of the lower molars. Many of these enamel-defects are so slight as to require the use of high-power objectives to discover them,[1] and yet are sufficiently large to furnish lodgement for masses of bacteria.

There is another feature which calls for consideration: for some time after the full eruption of the teeth, fragments of the partially calcified structure known as Nasmyth's membrane are attached to the teeth and occupy the depths of pits and sulci; they, no doubt, furnish lodgement for ferments and fermentable material.

If gross or even minute defects in enamel leave the dentin exposed at some point or points, the carious process is correspondingly favored. There are differences in the structure of the dentin which influence the rapidity and nature of the carious process. The existence of intra-globular spaces permits the quick progress of the disease. Differences of anatomical organization of the dentin itself also appear to affect the resistance offered by this tissue.

Chemical analysis of the calcium constituents of dentin, enamel, cementum, and bone shows the mineral basis of these tissues to be probably a phosphato-carbonate of calcium $(PO_4)_6Ca_{10}CO_3$ or $3((PO_4)_2Ca_3)$-$CaCO_3$; that is, saturated calcium phosphat-carbonate in a combination which corresponds to apatite $(PO_4)_6Ca_{10}Fl_2$.[2] This mineral substance and dentin and enamel, all show different rates of solubility in dilute acids, the mineral substance being most soluble. In animal tissues the calcium salts are combined with an albuminous bases; the nature of the union and the chemical composition of the "calcium albuminate" have not been made out. Williams[3] has shown that in the enamel there are two substances which differ in relative solubilities; hence it is a natural inference that although both may have the same

[1] Williams, *Dental Cosmos*, 1897. [2] Miller. [3] *Dental Cosmos*, 1895.

chemical composition they differ as to the molecular arrangement of their constituents.

The human teeth, as shown by Black,[1] do not exhibit sufficient variations in chemical composition to account for the differences observed in their decalcification during dental caries. As emphasized, "the variations in the amounts of salts in the dentin are not enough to explain their variations in hardness."[2] The hardness and solubility of dentin not being governed by the amount of calcium salts present, the differences of solubility must be looked for in the nature of the union between the calcium salts and the albuminous base; that is, it is a question of anatomical organization of the formed product of the dental tissues. "In the case of a chemical union between the organic and inorganic constituents of a tooth we should expect to find the dentin hard or soft according as the union is firm or unstable."[3]

The hardness or softness of teeth, the amount of calcium salts they contain, or even their anatomical alterations, cannot, as shown by Black, prevent the advent or progress of dental caries, although they undoubtedly do modify its character and rate of progress. The forces of attack being equal, a poorly organized and badly formed tooth will succumb sooner than one perfectly formed and of completely organized tissues; this law is constant in all biology. Of course, such gross malformations as areas of dentin marred by lack of enamel-formation—honeycombed teeth—offer an inviting field for dental caries; and yet in some cases such spaces may permanently escape caries. If the active causes of dental caries fail to assert themselves, the predisposing causes signify little or nothing. However, as the active causes of dental caries are of almost universal distribution, it is rare that such tissue as described escapes.

Faults of Form.—The outward forms of the teeth determine in a marked degree their vulnerability to dental caries, viewed both as to the separate forms, a lesser consideration, and, secondly, to the influence that form exercises upon the contact relationships of the teeth.

Teeth of faultless histological structure may invite the carious process if the pits and depressions which lie between the cusps are of unusual depth. Teeth of this description are, as a rule, broad upon the occlusal faces as compared with their cervical widths; hence in the dental arch, if the arrangement be normal, each tooth is separated from its fellow at the neck by a V-shaped interspace. Provided such teeth do not acquire roughened enamel-surfaces from the action of acids, locally generated, or administered medicinally, these spaces are generally kept free by the fluids of the mouth, which flow freely between the buccal and lingual cavities. If, however, the molar teeth are cuboidal

[1] *Dental Cosmos*, 1897. [2] Miller. [3] Miller.

in form, the bicuspids laterally flattened cylinders, and the incisors and cuspids rectangular in section, the approximal spaces have not the V-shaped and natural self-cleansing forms, but exhibit broad contact-surfaces, between which débris collects and is not removed by the saliva and the movements of the lips, cheeks, and tongue. The fibrous stems of vegetables and meat-fibres also tend to free the teeth from foreign matters; raw vegetables, such as celery, and tough, fibrous meats perform a useful service in this direction.

Arrangement of the Teeth.—No matter what the forms and structure of the teeth, if they be arranged in such a manner that uncleansible spaces are formed between adjoining teeth, a predisposition to caries is created. When, through the loss of a tooth, adjoining teeth so alter their positions that the natural cleansing agencies of the mouth are inactive, predisposition to caries is established. Teeth which have lost their antagonists are particularly prone to accumulate débris. This is marked in cases where several teeth, as the lower molars, have been lost; their antagonists become the seat of food-débris deposit and of salivary calculi, and sooner or later suffer from degeneration of the pericementum.

Other Local Predisposing Causes.—The relative position of the anterior borders of the masseter muscle with the teeth, is in some individuals far forward, forming about the buccal surfaces of both upper and lower third molars a cavity partially marked off from the general buccal cavity. These spaces are but little affected by the irrigation of the saliva, and are out of reach of the tip of the tongue; hence food-débris is likely to collect along the cervico-buccal margins of the teeth.

If the frænum of the tongue has its attachment abnormally near the tip, the organ is limited in its normal office; its sweep is not sufficiently free, so that not only is there difficulty in mastication, but a lessening of tongue-cleansing of the teeth, a double cause for the accumulation of débris.

Alterations in the character of the salivary and oral secretions may predispose to caries. It is questionable whether the saliva itself, as contained in the acini of the salivary glands, is ever of acid reaction, but it is certain that it is usually acid in reaction as found in the oral and buccal cavities. The mucous glands of the lips in conditions of irritation produce an acid secretion. The general distribution of acid in the fluids of the mouth would first exercise an influence as a general decalcifying agent, producing roughness or at least loss of polish of the enamel-surfaces, which would invite caries by affording lodgement for food-débris. In mouths showing an acid reaction of the saliva it is common to find a ropy appearance of the fluid, particu-

larly on the floor of the mouth. Mucin is precipitated by lactic acid, presumably the offending substance in acid saliva, and while no doubt the part played by the coagulated or partially coagulated mucin is in the formation of calculous deposits, yet coagula aid in the retention of food-débris. In the same manner, catarrhal conditions of the mouth have a part in the production of caries. In these cases there is an increased secretion of mucin, which acts when coagulated in the manner stated.

Clinical History of Caries.

The clinical history of dental caries records the observable phenomena associated with its inception, progress, and termination.

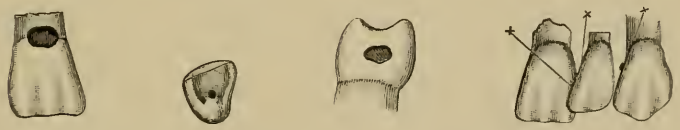
INCEPTION OF CARIES.

Caries usually begins upon the occlusal surfaces of the molar teeth, because here are usually found those defects of structure named under the head of predisposing causes of caries. In general terms, caries begins in situations which afford the most marked predisposition to it.

Fig. 227. Fig. 228. Fig. 229. Fig. 230. Fig. 231.

Fig. 232. Fig. 233. Fig. 234. Fig. 235. Fig. 236.

Fig. 237. Fig. 238. Fig. 239. Fig. 240.



"The situations in which caries appears are conveniently divided under four heads: (1) pits, grooves, and fissures of the enamel; (2) proximal surfaces; (3) smooth surfaces which from any cause are habitually unclean; (4) necks of the teeth, at or near the junction of the cement or enamel" (Black).

Caries appears more frequently in the permanent first molars than in any other teeth. Erupted at the ages of from five and one-half to seven and one-half years, these teeth are in position before any of the temporary teeth are lost; they are longer subjected to the attacks of

caries-producing conditions, are present before the patient has learned to voluntarily care for the teeth, and, in addition, usually present enamel-faults or configurations which aid in the retention of food-débris. At this period it is unusual for the dietary to contain the substances which act as mechanical cleansers, or for the child to masticate with sufficient vigor to aid this end. The lower first molars are usually affected before the upper, the latter showing caries at a later period (Figs. 227 and 228).

The upper incisor teeth, erupted next, do not, as a rule, exhibit marked evidences of caries so soon as the permanent second molars, erupted years after, although they are usually affected before the bicuspids. The lower anterior teeth are the last of all to be affected, and it is common to see the six lower anterior teeth free from caries years after all of the other teeth have been lost. This is attributable to the constant washing these teeth receive from the saliva and to the mechanical effects of tongue- and lip-movement and mastication.

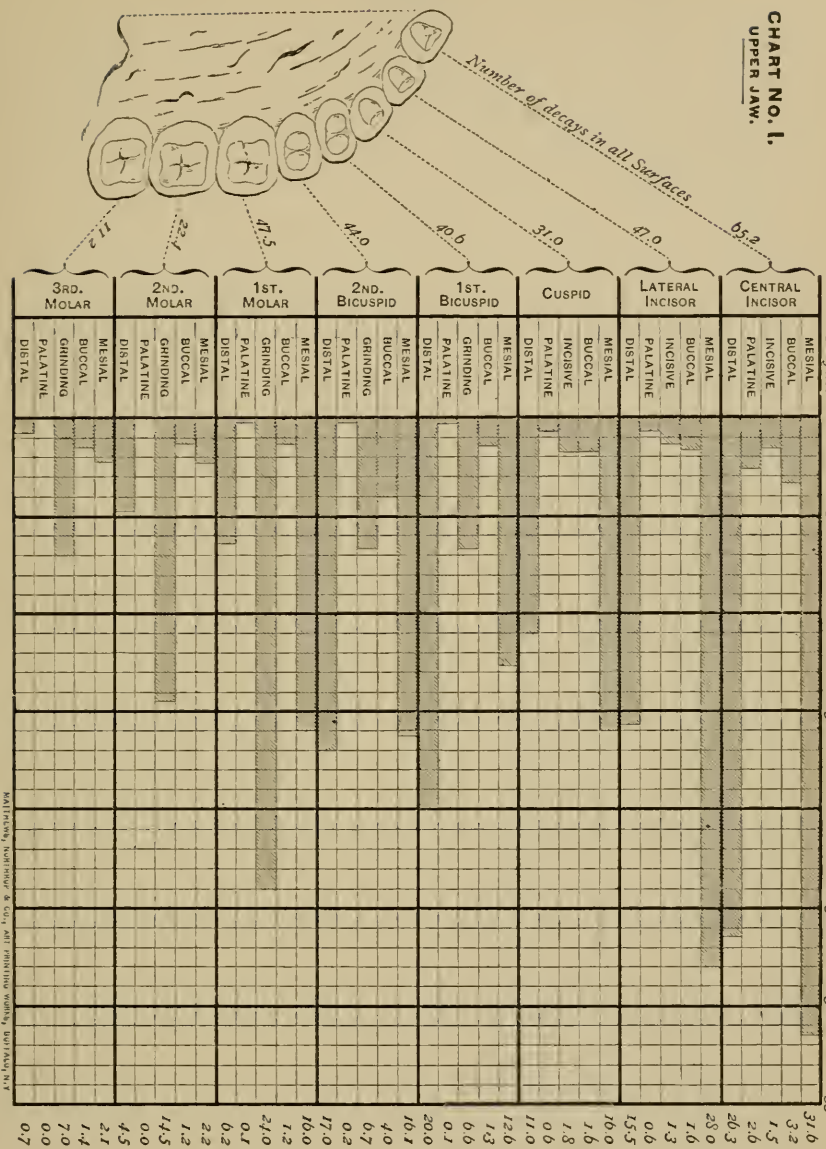
If a deep basilar pit exist, as it frequently does, upon the lingual surface of the upper lateral incisor (Fig. 238), it marks this tooth for an early victim. The approximal surfaces of the incisors offer inviting conditions. It will be remembered that the disto-approximal walls of the upper incisors are much more rounded than the mesio-approximal walls; hence these flattened surfaces afford a more ready lodgement for débris than the others. In the upper lateral incisors the mesial wall has frequently a depressed form, inviting the beginning of caries (Fig. 240).

The upper bicuspid teeth are next in point of vulnerability. If enamel-fissures exist upon the occlusal surfaces, they become the seat of the disease (Fig. 231); the pits marking the sulci-extremities of these teeth usually exhibit caries first. Even in the absence of these defects, the upper bicuspids are affected next in point of frequency, the nature of the contact between the bicuspids, and between bicuspids and first molars furnishing the lodgement for débris.

It is usual to find caries affect the distal surface of the upper central incisor some time after the disease makes its appearance on the mesial surface of the adjoining lateral incisor (Figs. 235 and 236). For a period the rounded surface is kept clean, but rarely escapes infection for long.

The mesial walls of the first molars are affected next in point of frequency, particularly if the distal wall of the second bicuspid have previously become carious. If the occlusal faces of the lower bicuspids have defects of structure—pits, or in the second bicuspids fissures—they become affected (Fig. 232); but in point of usual occurrence the mesial surfaces of the cuspids appear to succumb next. The somewhat flattened surface in contact with the rounded distal wall of the lateral incisor may invite the condition, or infection may occur after the appearance of caries in the

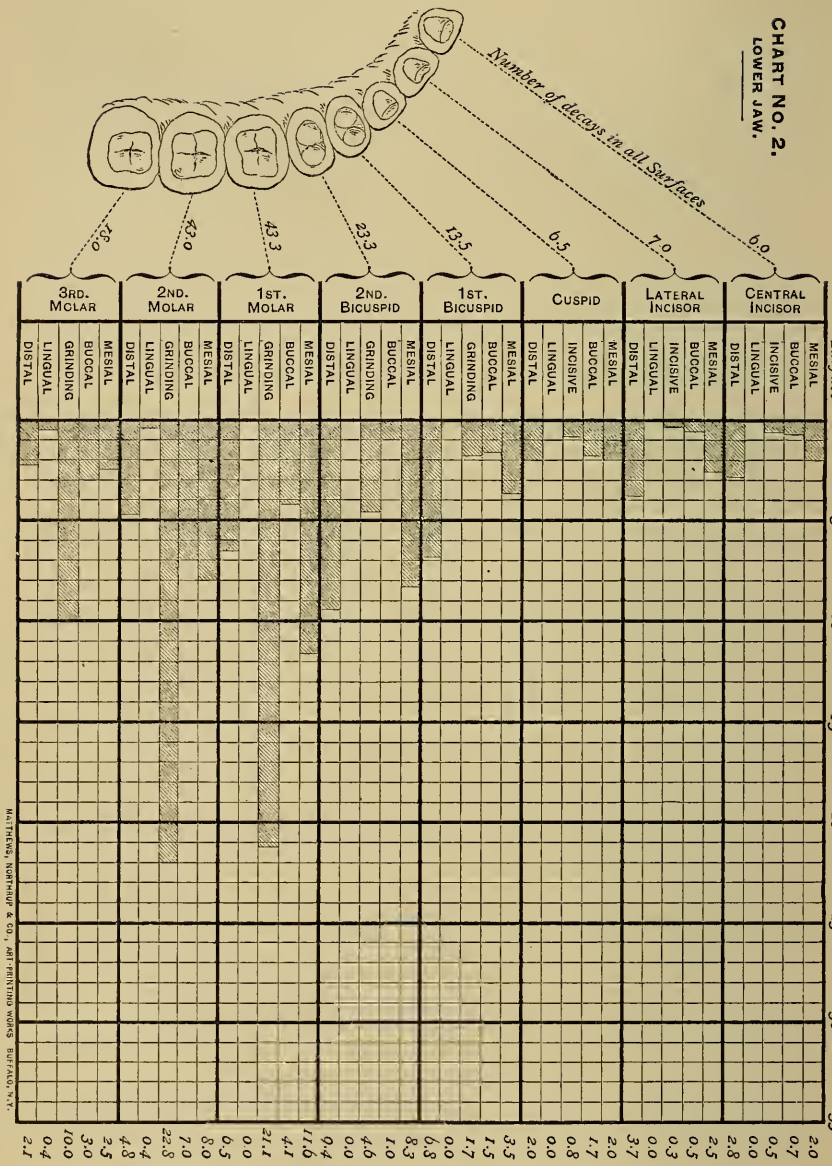
FIG. 241.



DESCRIPTION OF CHARTS.

These charts represent the number of carious cavities observed in *one hundred persons*, and the position of these cavities on the individual surfaces of the teeth. There are five columns of squares devoted to each tooth of one side of the mouth, representing the five surfaces as shown on the left hand. The number of cavities in the surface represented is shown by the number of squares darkened, so that the effect of the diagram as a whole gives a striking picture of the frequency of decay in the individual surfaces of the several teeth. On the right the percentage, or the number per hundred persons, is given in figures calculated to the first decimal point. On the left the percentage of cavities in the individual teeth for all surfaces is given in the same way. The cavities

FIG. 242.



occurring on one side of the mouth only are represented. And only one decay in an individual surface is counted; that is, if two or more pits are found decayed in the grinding surfaee of a molar, but one is counted; and the same rule is followed with all of the surfaees.

Charts No. 1 and 2 (upper and lower jaw) are made up from my records of fillings for 628 persons of all ages, and therefore represent what is seen in practice rather than the actual number that may oceur,

Charts No. 3 and 4 (upper and lower jaw) are made from 100 of my own patients between the ages of ten and twenty-five years, for whom I have filled all cavities and know the condition at present. They represent the actual number of eases in which the individual surfaces have decayed in these 100 persons. (Black.)

FIG. 243.

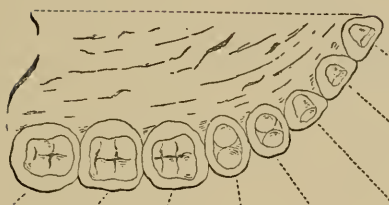


CHART NO. 3.
UPPER JAW.

Surface	Central Incisor	Lateral Incisor	Cuspid	1st Bicuspid	2nd Bicuspid	1st Molar	2nd Molar	3rd Molar
Mesial	3.5	7.5	6.5	8.5	7.0	6.0	10.5	4.5
Buccal	3.5	2.5	3.5	3.5	9.0	1.5	5.5	1.5
Incisive	.5	3.0	.0					
Palatine	5.0	9.0	.0	.0	.0	24.5	1.5	.0
Distal	17.5	28.5	3.5	14.0	6.0	6.0	5.0	.0
	33.0							

MATTHEWS, NORTHRUP & CO., ART-PRINTING WORKS, BUFFALO, N.Y.

FIG. 244.

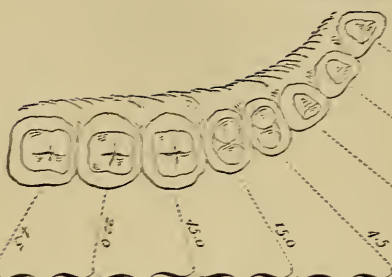


CHART No. 4.
LOWER JAW.

Surface	Central Incisor	Lateral Incisor	Cuspid	1st. Bicuspid	2nd. Bicuspid	1st. Molar	2nd. Molar	3rd. Molar
	Mesial	Mesial	Mesial	Mesial	Mesial	Mesial	Mesial	Mesial
	Buccal	Buccal	Buccal	Buccal	Buccal	Buccal	Buccal	Buccal
	Incisive	Incisive	Incisive	Grinding	Grinding	Grinding	Grinding	Grinding
	Lingual	Lingual	Lingual	Lingual	Lingual	Lingual	Lingual	Lingual
	Distal	Distal	Distal	Distal	Distal	Distal	Distal	Distal
5	.0	.0	.0	1.0	4.0	5.5	3.5	.0
10	.0	.0	.0	1.0	5.0	8.0	5.5	.0
15	.0	.0	.0	1.0	5.0	8.0	5.5	.0
20	.0	.0	.0	.0	5.5	8.0	5.5	.0
25	.0	.0	.0	.0	5.5	8.0	5.5	.0
30	.0	.0	.0	.0	5.5	8.0	5.5	.0
35	.0	.0	.0	.0	5.5	8.0	5.5	.0
	1.0	1.5	2.0	1.5	5.5	3.5	2.5	.0
	.0	.0	.0	1.0	4.0	3.5	2.5	.0
	.0	.0	.0	1.0	5.0	3.5	2.5	.0
	.0	.0	.0	.0	5.0	3.5	2.5	.0
	.0	.0	.0	.0	5.0	3.5	2.5	.0
	.0	.0	.0	.0	5.0	3.5	2.5	.0
	.0	.0	.0	.0	5.0	3.5	2.5	.0
	3.0	3.5	5.5	4.5	15.0	15.0	12.0	3.5

Matthews, Northrup & Co. Art Printing Works, Buffalo, N.Y.

latter tooth. When the approximal face of one tooth becomes affected by caries it is usual to find the adjoining tooth also affected after a period, a natural inference when the etiology of caries is remembered.

It is unusual for the third molars, upper or lower, to be free from caries many years after their eruption. Although the teeth are usually affected in about the order given, it is to be remembered that peculiarities of structure, form, position, and personal habits modify the occurrence and progress of caries.

The preceding charts represent records made by Black as to the relative frequency of dental caries. "First molars, being extracted in larger proportion than other teeth, the numbers given for these teeth in the tables are too low."

RAPIDITY OF PROGRESS.

All other conditions being equal, caries progresses most rapidly in situations where the persistent lodgement of food-débris and bacteria is

FIG. 245.

Section of human bicuspid, showing commencement of caries: *a* and a^1, appearances caused in enamel and dentin by the acid of decay; *b* and b^2, shreds of a felt-like mass of bacteria raised from the surface of the enamel; *c*, a cavity. × 12. (Williams.)

assured. Hence, it progresses most rapidly when deep fissures of molars have invited its beginnings, in deep pits, in approximal cavities when the tooth-walls are much flattened, and upon surfaces which are partially overlaid by the soft tissues in such manner that

the surfaces are not irrigated. Cases are frequently observed where the only external evidence of caries in a molar or bicuspid tooth is a blue-black line marking the fissure, and yet almost the entire dentin of the crown may be decalcified. Fig. 245 shows a bicuspid tooth from the mouth of a woman, aged thirty-five years, presenting no external evidence of caries, yet histological examination showed that decalcification had proceeded deeply into the substance of the dentin.[1] "There is no doubt that many cases of so-called soft teeth have their softness caused by conditions similar to this."[2]

While, as a result, the extent of dentinal invasion bears some relationship to the extent of enamel-loss, the above examples are sufficient in themselves to show that any preconceived opinions as to the extent of carious progress based upon the size of the external orifice are delusive. In point of fact, the most extensive decalcifications are usually associated with limited enamel-loss (Fig. 246, *A*).

The physical appearance of the carious cavity affords some indication of the depth of decalcification. A cavity having slightly discolored ragged enamel-edges, but filled with soft débris, will usually be found to be deeply affected. On the contrary, cavities with broad mouths, but little débris, and with discolored walls, are usually associated with limited decalcification (Fig. 246, *B*).

Approximal cavities in bicuspids and molars are more likely to be deep and extensive before discovery, than are approximal cavities in the incisors: the reason is obvious.

While, as a rule, caries is a regularly progressive disease, going deeper and deeper until all of the tooth-crown is destroyed, conditions occur which modify its progress. If the cavity-walls be broken away in such manner as to permit a free circulation of saliva in the cavity and the tendency to débris-accumulation is thus lessened (Fig. 246, *B*), the progress of the disease may be retarded. In some cases, indeed, it is permanently checked. The latter phenomenon, however, is rarely observable except in teeth of a high grade of organization.

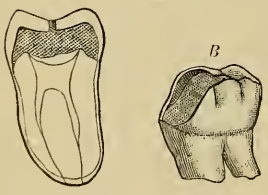


FIG. 246.

Again, caries may progress rapidly for a period, and then receive a check to its progress. Teeth previously free from the disease may suddenly fall victims to its rapid and widespread progress. No doubt, in many of these cases there are removed from or added to the local oral conditions constitutional influences which deter or favor the local development of caries-producing bacteria.

While caries appears at all ages from childhood to old age, its rav-

[1] Williams. [2] Black.

ages are most pronounced and its progress most rapid during the period of adolescence and early maturity. Its effects are most marked between the ages of eight and twenty-five years. As a rule, a denture which remains for twenty-five years unaffected by caries remains unaffected or but slightly affected to an indefinite age. To be sure, this implies two conditions: first, that the active causes of caries have been in but slight evidence; and, secondly, that the denture is of the highest order. The classes of denture which escape are, perfectly formed and symmetrically arranged teeth, in the mouths of patients who lead sanitary lives, who masticate vigorously, and who escape other diseases.

Caries beginning at the junction of the cementum and enamel of the teeth has a somewhat different clinical history from that noted when its occurrence is in other situations. Its progress is subject to great variations. In any of the catarrhal conditions or atrophic conditions of the gum which lie bare the neck-cementum caries usually occurs. It occurs also as a process secondary to mechanical abrasion and erosion of the teeth. Teeth affected by erosion, however, as has been pointed out, are commonly exempt from dental caries.

CHAPTER XIV.

DENTAL CARIES: PATHOLOGY AND MORBID ANATOMY.

THE pathology of dental caries deals with the *modus operandi* of the agencies which bring about the dissolution of the calcic tissues of the teeth. The morbid anatomy describes the changes of structure which these tissues undergo in the course of the dissolution.

The pathology of the disease varies according to the method of attack. If a break or exposure in the enamel permits access of the

FIG. 247.

Sections of normal human enamel, showing thick, felt-like mass of micro-organisms slightly raised from the surface of the tissue by pressure of the cover-glass in mounting. ×350. (Williams.)

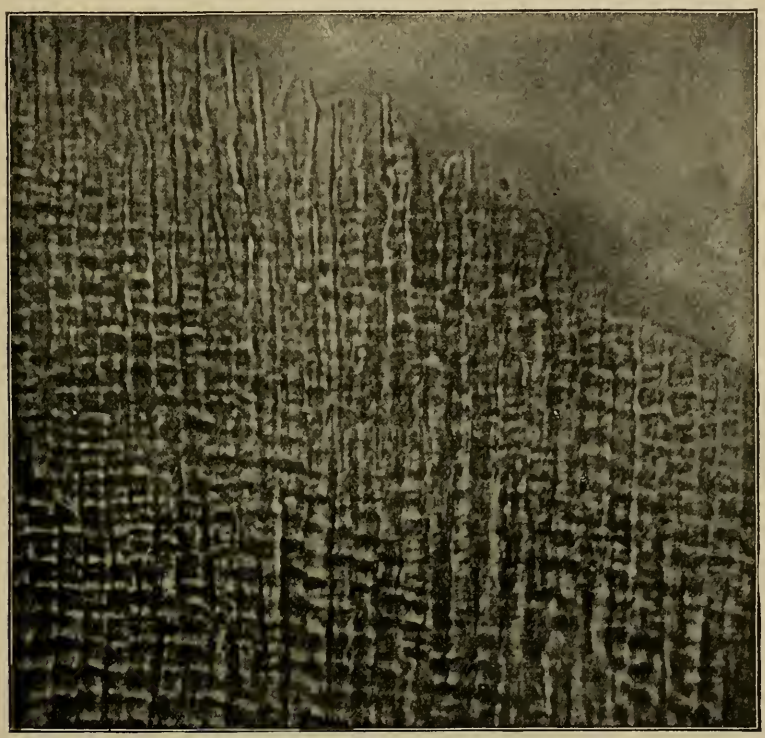
active causes of caries to the dentin, the phenomena of dentinal caries begin at once; if, however, the dentin is completely sheathed by unbroken enamel, dissolution of the enamel must occur before caries of the dentin begins. While the essential nature of enamel and dentinal

caries is nearly alike, there are distinctive differences in the modes in which their dissolution is brought about; it should also be noted that caries of cementum exhibits anatomical peculiarities.

Caries of the Enamel.

For a clear understanding of caries of the enamel it is necessary to recall the mode of enamel-formation and its composition, which were

Fig. 248.



Section of carious enamel: bacteria removed to show action of acid on enamel-rods. ×550. (Williams.)

discovered and set forth by J. L. Williams, who furnished also the first accurate description of the early and later phenomena of enamel caries.[1]

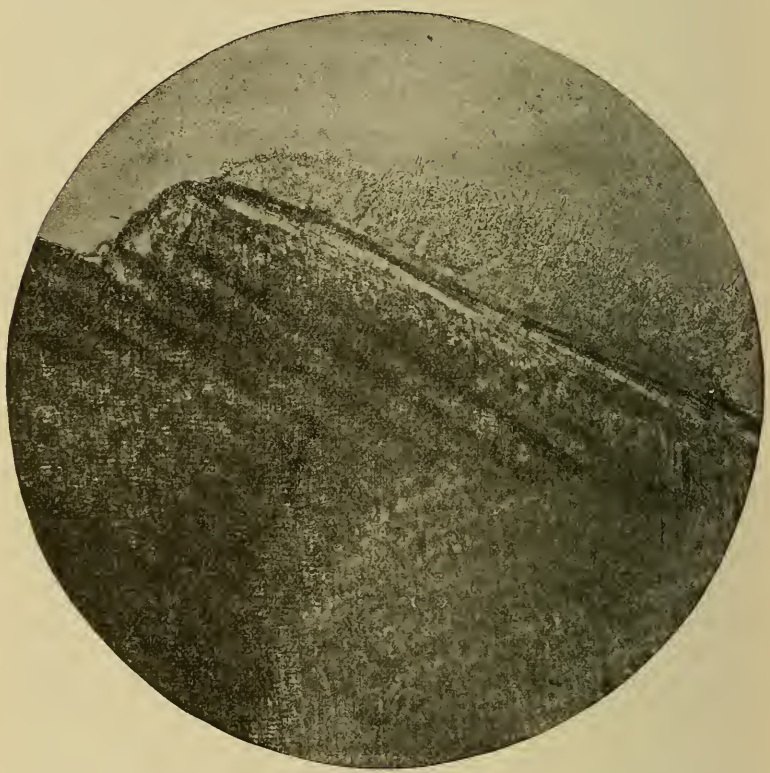
The enamel is composed of two different substances, cementing-sub-

[1] It is but just to state in this connection that Miller's descriptions of the phenomena and pathology and morbid anatomy of dental caries (*Micro-organisms of the Human Mouth*) were as complete and full as the contemporary knowledge of dental histology could make them; even more, his observations, correctly interpreted, anticipated some of the discoveries of Williams. Miller's descriptions of caries of the enamel given before 1890 would answer, with a few modifications, for a description of our present knowledge of the subject. This in no way belittles the work of Williams, which must stand as a permanent monument to his acumen, patience, and skill in histological technique.

stance and globules of uniform size, which are built upon one another until rod-like forms, the enamel-prisms, are produced. Each of these globules and each rod are bound together by calcified cement-substance. Acted upon by dilute acids, the cementing-substance is seen to be more soluble than the globular bodies.

Williams has shown that felt-like adherent masses of bacteria may find lodgement in the remains of Nasmyth's membrane, in such situa-

Fig. 249.



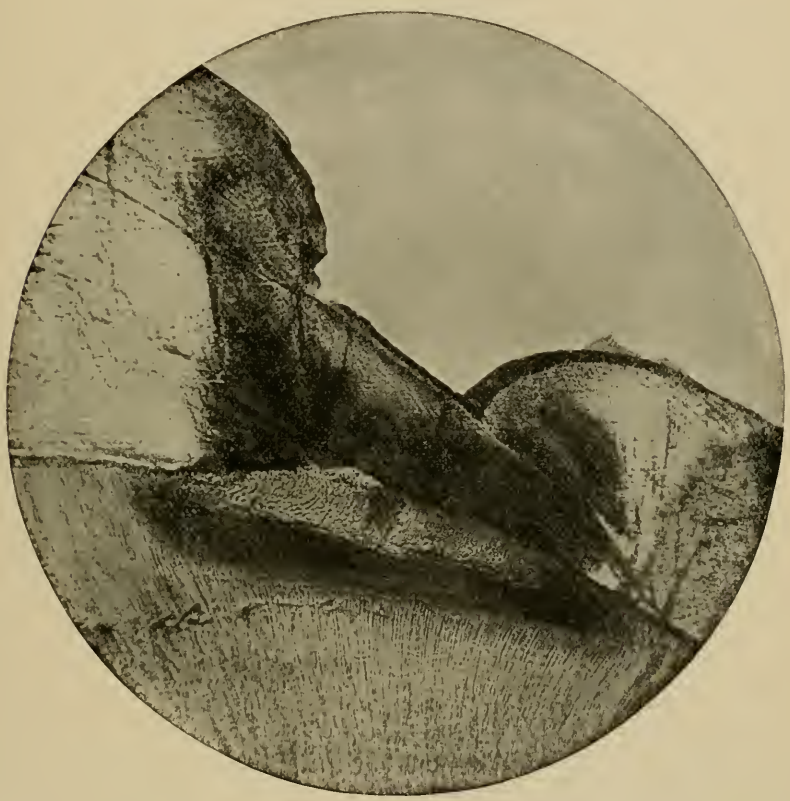
Section of human enamel, approximal surface of incisor, showing micro-organisms of decay attached to the surface and marked effect upon tissue caused by penetration of acid excreted by the bacteria. × 250. (Williams.)

tions as the approximal surfaces of the teeth (Fig. 247). "Lining the cavities or covering the surface where decay has commenced there is always to be seen a thick, felt-like mass of acid-forming micro-organisms. *This mass of fungi is so dense and adhesive as to make it highly improbable that the enamel is affected, except in rare or special instances, by any acid other than that which is being excreted by the bacteria at the very point where they are attached to the enamel.* This thick, glutinous-like

mass of fungi also prevents the excreted acid from being washed away, so that it exerts its full chemical power upon calcific tissue."

When viewed in sections, the appearances produced by the action of dilute lactic acid upon the enamel are similar whether the decalcification be artificial or due to the progress of caries. The cement-substance, being more soluble than the calcified globules, is dissolved first, which

FIG. 250.



Section of human enamel, approximal surface of molar, showing decay temporarily arrested by a line of stratification; micro-organisms deeply stained. × 150. (Williams.)

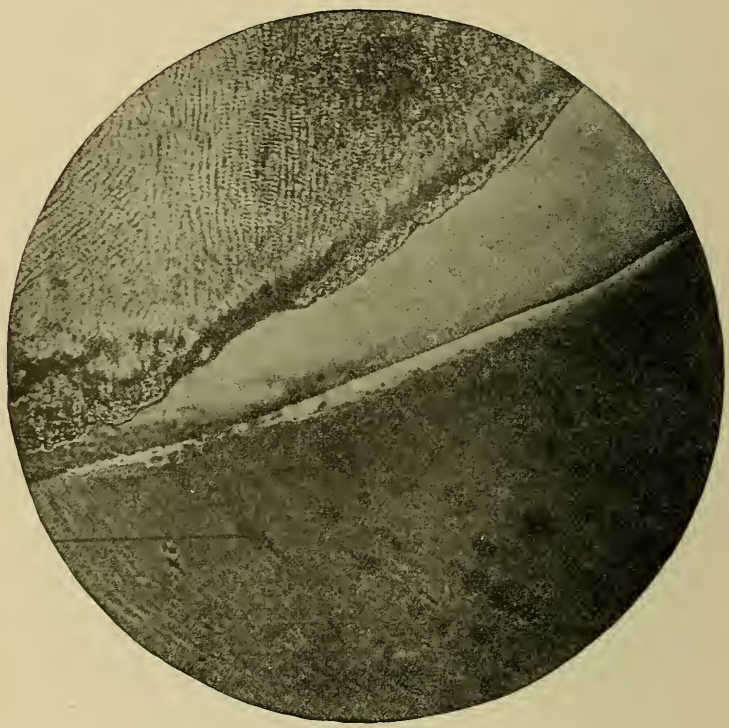
forms an irregular, roughened surface at the point of attack. The gradual dissolution of the cement-substance brings into relief the structure of the enamel-rods (Fig. 248). The gradual loss of cement-substance unbinds the enamel-globules, which are in turn dissolved and washed away, leaving a depression or cavity.

It is common to find in the early stages of enamel-caries and cavity-formation a discoloration of the affected tissue in those cases where the disease progresses very slowly. Whether this discoloration is caused by

the formation of a sulfid,[1] or is due to the action of chromogenic bacteria, has not been made out.

In Fig. 249 it is seen that the advance of decalcification has brought into bold relief the normal pigmented lines of the enamel (the striæ of Retzius), with the apparent indication that these lines offer territories of increased resistance to decalcification (see Erosion). Lines of

Fig. 251.



Section of carious tooth, showing appearances of decay in enamel and dentin at the line of union of these tissues; the dark spots shown in the enamel and dentin at *a* and *b* are masses of micro-organisms. × 250. (Williams.)

enamel-stratification may temporarily arrest the direct progress of enamel-dissolution (Fig. 250). Decalcification may proceed to comparatively great depth before cavity-formation occurs. In fact, the decalcification may penetrate the entire thickness of the enamel and dentin before there is breaking down of the enamel-walls. When the entire thickness of the enamel is penetrated and the dentin attacked there is a change in the mode of progress of the decalcification. It will be recalled that the first deposited layers of enamel are made in a layer of cement-substance next to the dentin; this sheet of material appears

[1] Black.

to offer less resistance to decalcification than other portions of the enamel.

If the carious process begin in a fissure or pit, there is little or no tissue-loss necessary before the beginning of this secondary phase of enamel-caries. The solution of this interzonal layer of enamel occurs promptly, and decalcification proceeds along the enamel-rods from the dentin side with increased rapidity (Fig. 251). Bacteria growing in the

FIG. 252.

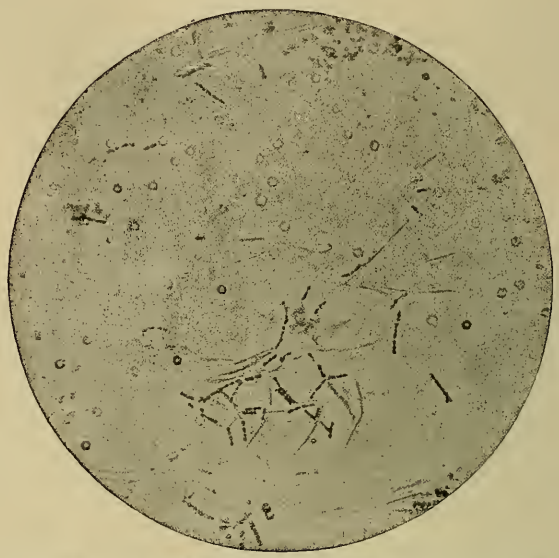
Cover-glass preparation from scrapings of white, opaque decaying enamel: the cement-substance between the rods is seen to be dissolved away, and the crevasses thus formed are filled with round and oval forms of micrococci and bacteria. Stained by the Gram method. ×450. (Williams.)

spaces from which interprismatic cement-substance has disappeared cause detachment of masses of partially decalcified rods (Fig. 252).

In the ultimate breaking down of the enamel the rods first separate; the outlines of the several globules of which the rods are composed are brought into plain view; next, the calcified plasmic strings noted in enamel-formation become evident; and finally the bead-like masses upon these strings are left as the ultimate granular detritus of the enamel.

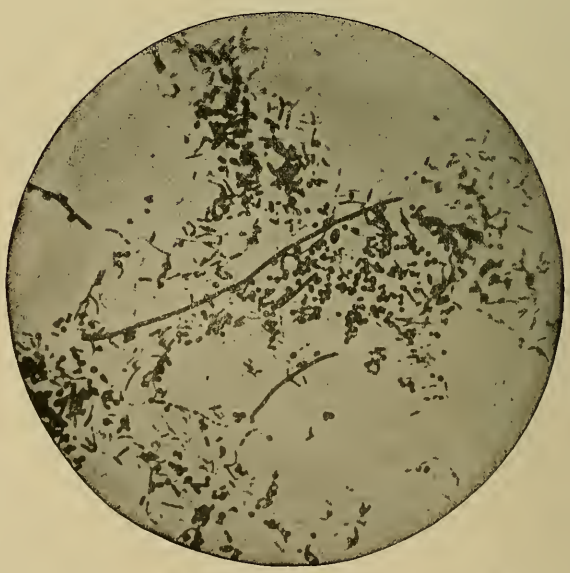
In cases of rapid enamel-dissolution Williams found streptococci almost invariably present; and suggests tentatively that the variety of

FIG. 253.



A form of streptococcus found abundantly in mouths where very rapid decay of the teeth is in progress. ×750. (Williams.)

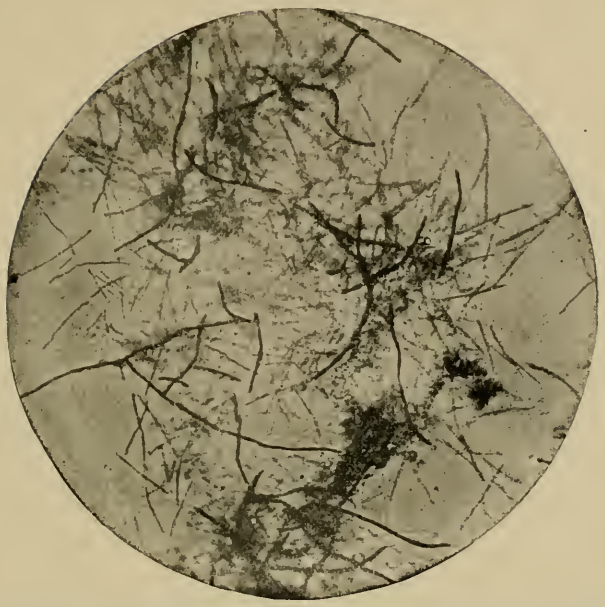
FIG. 254.



Various forms of micrococci and bacteria from decaying enamel. Photographed by Mr. Andrew Pringle from Williams' cover-glass preparation. ×1000. (Williams.)

organisms may be the factor governing the rapidity of dissolution (Figs.

FIG. 255.



Cover-glass preparation of scrapings from decay of enamel: shows leptothrix buccalis maxima and bacillus buccalis maximus of Miller. Stained by Gram method. × 850. (Williams.)

FIG. 256.

Scrapings of micro-organisms from the approximal surface of a decaying tooth: shows the leptothrix buccalis maxima and the bacillus buccalis maximus of Miller. × 1500. (Williams.)

253 and 254). The large cocci and diplococci shown in Fig. 252 were always found in the backward decay of enamel.

"In the direct caries of enamel the cavities are lined with leptothrix and thread-like forms" (Fig. 255).

"The leptothrix buccalis maxima and the bacillus buccalis maximus of Miller are nearly always found, the latter more sparingly" (Fig. 256).

"Beneath the felt-like masses of thread-forms and lying in contact with the decomposing enamel in direct decay, and also in deep cracks and fissures in backward decay, there is invariably found a short, thick bacillus, usually constricted in the centre."

Caries of Dentin.

Sections of carious dentin show the evidences of several processes—chemical, physiological, and pathological—in operation at the same time (Fig. 257). First, the enamel is broken down about a fissure, as shown in the illustration, or is dissolved from a portion of one of the tooth-walls (also shown). The margins of the mouth of the cavity consist of decalcified and discolored enamel. Between the floor of this cavity and the pulp-chamber are several distinct zones, each having a definite pathological significance. The outermost zones are soft and discolored, but lying beyond them next to the pulp is a translucent field, named, from the investigator who first called attention to it, the transparent zone of Tomes. In the illustration it will be noted that the transparent zone is continuous with the projection of a secondary deposit upon a portion of the pulp-chamber wall.

Upon gross examination it will be seen that for some distance from the mouth of the cavity and in the dentin beneath the enamel, decalcification has occurred, and that the formerly hard tissue has now but a cartilaginous density; it is soft to cutting-instruments. After washing the cavity the fluid which can be squeezed out of the dentin is invariably acid in reaction. The more rapid destruction of dentin causes an undermining of the enamel, which, being deprived of its normal support, suffers more or less fracture about its margins.

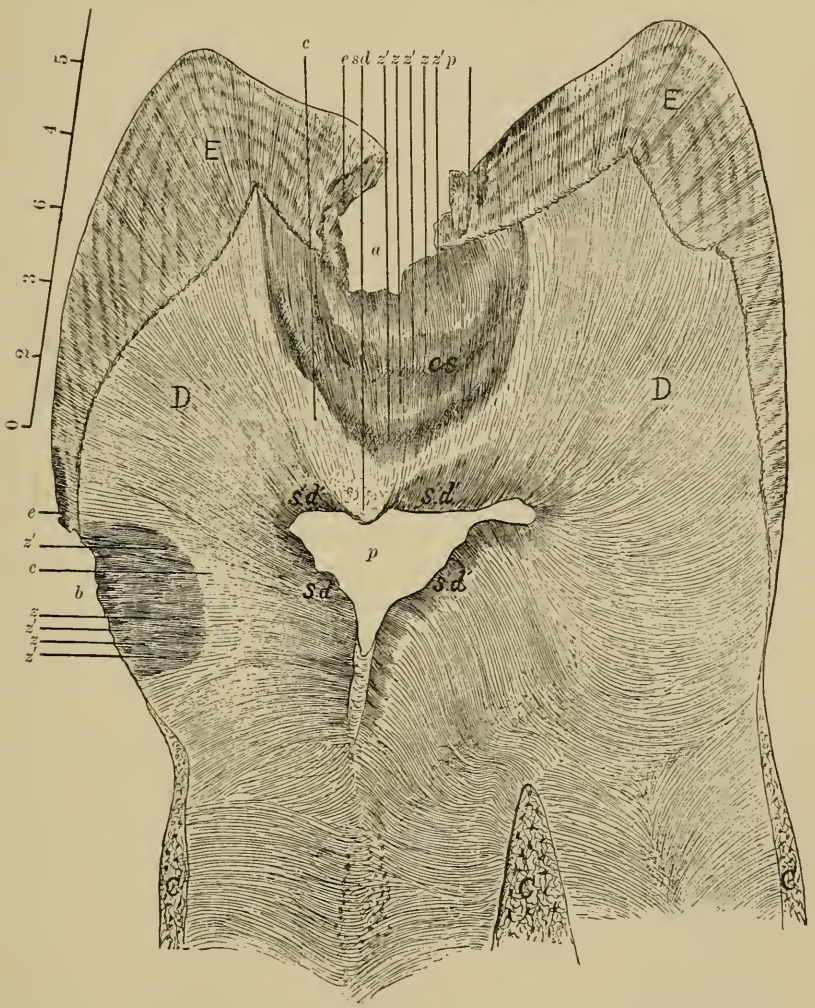
"Sections made of carious dentin parallel with the direction of the dentinal tubuli, and stained with fuchsin, show that the superficial layers of the softened dentin are filled with bacterial forms; the deeper layers of *decalcified* dentin are not infected. The decalcification precedes the invasion of the bacteria themselves into the dentinal tubuli."[1]

How long the protoplasmic processes in the dentin retain their vitality when the dentin about them has been decalcified has not been determined. It would appear, however, from clinical tests that they do maintain their vitality for some time. Softened dentin is

[1] Miller.

often exquisitely sensitive to touch; whether due to direct contact of the instrument with the dentinal processes, or due to the pressure exerted being transmitted to the vital processes beyond, is not

FIG. 257.



Longitudinal ground-section through the crown of an inferior molar of the negro: *E*, enamel; *D*, dentin; *C*, cement; *p*, pulp-chamber; *a*, large decay, from the grinding surface; *b*, small decay, from the mesial surface; *cs*, cone of septic invasion and discoloration; *e*, partially decalcified and discolored enamel around the carious cavity; *z*, dark cones; *z'*, clearer cones; *z'p*, oldest cones where putrefaction of the tooth cartilage begins; *c*, outer transparent zone, or zone of Tomes; *sd*, secondary dentin, caused by irritation; *s'd'*, secondary dentin deposited by normal physiological process, recession of the pulp. This figure is drawn from a ground and polished section mounted in Canada balsam. (Gysi.)

known. Upon this point depends whether a dentin-matrix which has once undergone any degree of decalcification can ever be the seat of reconstructive activity. Certainly, in the absence of live protoplasm

in it, softened dentin must remain soft. Clinical records appear to indicate that softened dentin has, after a long period of perfect protection, regained its hardness. This change can only be brought about through the agency of the odontoblastic processes, and very probably cannot extend beyond the boundaries of the sheaths of Neumann. Histological data are wanting in this connection.

It is clearly shown that the primary ferment in dental caries performs one office: it causes removal of the calcium salts of the dentin in advance of the organisms. What effect it may have upon the organic structures remaining after complete decalcification, is questionable. It may or may not produce alterations in the gelatinous matrix which are a necessary preliminary to its future dissolution. Subsequent to decalcification is the destruction of the organic matrix, which is broken down through the agency of peptonizing bacteria. It will be observed that the peptonizing ferment which causes this dissolution acts in the presence of an acid.

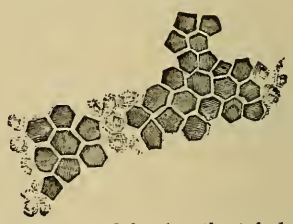
The invasion of organisms takes place *via* the dentinal tubuli. In the deeper portions of tubules micrococci appear to predominate over the rod-forms, which are also present; although one tubule may be filled with cocci and its neighbor with rod-forms (Fig. 258). It is only in the more superficial layers that the thread-forms are found in numbers.

Fig. 258.

Decayed dentin, showing a mixed infection with cocci and bacilli. × 400. (Miller.)

The invasion of the tubules is followed by their dilatation. This change is more plainly seen in sections cut at right angles to the axes of the tubules; they are distended to several times their usual size, until there is an entire disappearance of the intertubular tissue (Fig. 259). The walls of the sheaths of Neumann undergo thickening, this structure maintaining its form to the last stage of caries, and acquiring the tobacco-stem appearance described by Tomes.

Fig. 259.



Cross-section of decayed dentin: the tubules through reciprocal pressure have assumed the shape of 5–6 sided prisms. (Miller.)

Miller points out "that the thickening of the sheath is not a vital process, since it may be clearly observed in specimens of artificial decay. In the progress of caries the thickened tubes dilate; a contraction of the lumen is only found when it has occurred before softening began.

The tubes dilate before losing their identity as tubes—*i. e.*, they undergo softening. Regarding the sheaths of Neumann as transitional material, numerous data point to the conclusion that the calcium salts contained in them are held in looser bonds—*i. e.*, are more soluble than the calcium salts impregnating the intertubular dentin.

It was pointed out by Röse[1] (see Chapter VIII.) that what are called the transverse processes of the dentinal tubuli react to stains like the sheaths of Neumann. Williams[2] has shown that dentin is probably built up of globular bodies. Assuming that the process of calcification of dentin resembles that of enamel, and that both globules and an interglobular substance are deposited in a fibrillated matrix, the appearance of these transverse markings would be explained as interglobular substance becoming marked by a partial decalcification, and exhibiting a higher degree of solubility than the globular bodies. Röse,[3] by the use of the combined Koch[4] and Golgi methods of staining, found that these transverse branches of Neumann's sheaths do not contain protoplasmic offshoots from odontoblastic processes; they stain as solid masses. Therefore, the name transverse tubules is a misnomer, and some explanation other than tubule penetration must be found for the invasion of micro-organisms along these paths.

TUBE-CASTS.

In the zone of decalcification in advance of bacterial invasion of the tubes are found rod-shaped bodies, first described by J. Tomes. They occur in both natural and *artificial* decay; hence it must be inferred that their presence is independent of vitality in the dentin—*i. e.*, they are not caused by a vital formative process. "The rods do not dissolve in organic acids; neither alcohol nor chloroform has any effect upon them; but the addition of dilute sulfuric acid causes their quick dissolution. In some cases loose fragments of these rods may be found surrounded by cocci, the rods probably having been formed before dilatation of the tubes and left loose in the tubules by the dilatation. The rods are brittle." These features indicated calcic formations, but the quantity of mineral was so small that the formation of calcium sulfate crystals could not be made out.[5]

FIG. 260.

[1] *Dental Cosmos*, 1893.
[2] *Ibid.*, 1896.
[3] *Ibid.*
[4] *Ibid.*
[5] Miller.

THE TRANSPARENT ZONE.

Far in advance of the zone of decalcification the dentin acquires a translucent appearance resembling senile dentin, and having a similar appearance to the dentin in some cases of abrasion and erosion. Miller's studies indicate positively that the translucent zone does not appear in caries of dead dentin, in artificial caries, nor in caries of human teeth mounted upon plates.

Tomes and Magitot both regarded the transparency as an attempt made by nature to impede the progress of caries. Walkhoff regards it as evidence of stimulation of a formative activity, causing the production of intercellular substance at the expense of the cells and primarily their offshoots.[1] Miller showed that there is rather an increase than diminution of the calcium salts in the transparent zone.

It will be noted that in Fig. 257 the transparent zone is continuous with a new dentin-formation upon the wall of the pulp-chamber. These evidences point to the truth of Walkhoff's explanation of the process, and indicate that the transparent appearance is the result of a vital reaction. The protoplasmic processes of the dentin being subjected to that degree of stimulation productive of formative activity, respond, and secondary deposits occur. While this may be, and no doubt is, a protective mechanism of nature, to assert that it is a specific provision for the arrest of caries is perhaps overstating the degree of cell-differentiation involved. Clinical experience indicates, however, that transparent dentin does offer increased resistance to the progress of caries.

Cavities in which caries has ceased spontaneously exhibit frequently the smooth, shining appearance noted in connection with mechanical abrasion; probably the sclerotic process noted above is the cause of the altered texture and appearance.

Caries of Cementum.

Decalcification appears to affect first the calcified rods known as Sharpey's fibres; the saprophytic fungi, following these paths, invade the cementum in a manner similar to dentin invasion. The bacteria invade the sites of cement-corpuscles and their offshoots.

Pigmentation in Caries.

In the more slowly progressing cases of caries more or less discoloration of dentin and surrounding enamel occurs. The colors range from yellow to jet-black. "*The intensity of the discoloration is in inverse proportion to the rapidity of the progress of the disease.*" The discolorations follow upon and do not precede the carious process. In limited, outlined areas

[1] Miller.

of enamel where the primary stage of enamel-caries has begun, discoloration may occur, and the carious process be checked. Whether there is any connection between the pigmentation and the cessation of caries is not known, although in many instances this would seem to be the case.

The chemical nature of these discolorations has not been made out; in the cases of deep enamel-staining they appear to be due to the deposit of precipitates upon the area of decalcification.

The discolorations of carious dentin may be due to the action of chromogenic bacteria. Miller isolated from the mouth an organism which he named bacillus fuscans, and "which, cultivated on the surface of nutritive agar-agar, imparts to the medium in a few weeks a yellowish-brown color, which gradually darkens and extends deeper into the substratum as the age of the culture increases."

The discolorations of dentin in teeth having vital pulps are probably not at all of similar origin to the discolorations (blue-black) of the dentin of pulpless teeth.

CHAPTER XV.

DENTAL CARIES: DIAGNOSIS, SYMPTOMS, AND PROGNOSIS.

The diagnosis of dental caries is made through objective signs and subjective symptoms. The signs are the existence of cavities and of softened areas, directly visible or made evident through instrumental means. The symptoms are pains of several degrees of intensity. The nature and intensity of the pains furnish a guide to the depth of the carious invasion, and but an indirect indication of the location of the disease.

Diagnosis by Objective Signs.

Caries is to be sought for in those situations where experience, as recorded in the clinical history of the disease, teaches the operator to examine. Many cavities, particularly those of large size, are evident at first glance; others require only the light of the mouth-mirror to show them plainly, but many require instrumental aid in the search. The enamel of the teeth should exhibit a hard, smooth, unbroken surface, and any part of its surface which will admit the point of a sharp exploring-instrument is defective, usually by reason of the presence of caries. The search for caries should be made systematically; and to insure thoroughness of record every discovered cavity should at the time of examination be recorded upon a suitable diagram.

Mouth-mirrors are required to reflect light upon surfaces of the teeth, large ones being used for this purpose, and smaller ones to reflect the images of invisible tooth-surfaces. Exploring-instruments of various forms are used, having sharp points and shafts bent in such manner that all of the surfaces of every tooth can be traversed by the instrument-points. Floss-silk is required to pass between the teeth to detect areas of roughness. Wedging-appliances are also used to press apart contiguous teeth sufficiently to admit the exploring-instruments. A general survey of each tooth is made, aided by reflected light, and any evident cavities are recorded.

In the absence of any directly visible cavities, an indication of their presence and depth may be secured by having the patient take a mouthful of cool—not cold—water, and, closing the mouth, to distend the cheeks; if there be cavities of any depth, the presence of the water will usually cause pain, indefinitely located. If pain be elicited, direct

search should be made in the approximal spaces of the side to which the pain is referred.

In the subsequent examination two systematic methods are pursued. In one method, the occlusal faces of all the teeth are first examined in one survey, then the interproximal spaces, and, lastly, the cervices of the teeth, and the margins of fillings, if there be any. In the other method, every portion of each tooth is examined, beginning with a central incisor or terminal molar, before passing to the adjoining tooth. The first method is more commonly followed.

Fissures and pits in the occlusal, buccal, or labial surfaces of the teeth which will admit and "catch" the points of fine exploring-instruments are defective; when present in the teeth of youth and young adults, they usually overlie carious dentin. Found in the teeth of middle-aged patients they may have little or no clinical significance. The basilar pits of the upper incisors require examination also. Approximal surfaces are first explored with exploring-points, which detect the presence of caries by catching the margin of a cavity or sinking into areas of softening. In the absence of evident cavities, some force should be applied to detect soft spots. Many spaces will not admit even fine exploring-instruments, in which cases waxed floss-silk may be passed between the teeth and drawn backward and forward; if it "frays," it indicates the existence of an area requiring attention. The use of wedges, immediate or slow, may be required for further examination of such spaces. The necks of the teeth should be examined with sharp points to note any softness of the tooth-tissues. The margins, particularly the cervical and neighboring margins, of every filling should be explored to test the integrity of the junction of filling and tooth.

Subjective Symptoms of Caries.

HYPERSENSITIVITY OF DENTIN.

The exposure of dentin to external agencies is so commonly followed by an increase in the normal sensitivity that the condition requires description in itself. It is a general condition attendant upon abrasion, erosion, and caries, and has a therapeutics of its own.

The term sensitive dentin applied to this condition is a misnomer; all vital dentin is sensitive, and its degree of sensitivity differs markedly in individuals; it is only when hypersensitivity is observed that the condition becomes pathological.

Hypersensitivity of dentin may be defined as such a degree of sensitiveness as interferes with the proper excavation and shaping of a carious cavity; or which, in the absence of dental ministrations, causes painful symptoms, as a rule reflected about neighboring parts.

Causes.—Normally the protoplasmic filaments of the dentin are completely protected from contact with external sources of irritation by the non-conducting layer of enamel which sheaths the crown of a tooth; it receives and, in all probability, notes stimuli due to changes in the temperature of the enamel which are induced by the introduction of hot or cold substances into the mouth. In general terms, the range of temperature of substances introduced into the mouth should be between about 50° F., the temperature of spring water, and 105° F. or slightly higher, the temperature of cooked foods. It is a common practice among the majority of persons to use ice water at a temperature of 32° to 35° F., and to have certain food-materials served very hot, at say 130° F. It is evident that the pulps of teeth and their terminals, the odontoblastic processes in the dentin, subjected to these abnormal stimuli must suffer according to the degree of the frequency and extent of the temperature-change—*i. e.*, the irritability of the parts is increased.

It is beyond doubt that individuals differ as to the degree of normal dentinal sensitivity; the dentin of one person may be cut freely without evidence of marked pain; in another, the touch of an instrument to the newly exposed dentin is productive of a paroxysm of pain. The difference in degree of irritability is manifested in another manner: if a mild sedative—for example, oil of cloves—be applied to the hypersensitive dentin of one person, it may remove the distressing symptoms, but with others it may be necessary to employ the most extreme measures to reduce in any degree the hypersensitivity.

As soon as an area of dentin loses its normal protective covering—the enamel, it is subjected to altered conditions, to sources of irritation from which it is normally free. In the majority of cases the exposure is brought about by the solution of overlying enamel by lactic acid. In others a fissure, a pit, or other malformation of the enamel may lay the dentin bare; or, as is frequently noted, the recession of the gum-margin may expose the thin layer of cementum covering the neck of a tooth, and this removed by abrasion exposes the surface of the dentin beneath. Mechanical abrasion of the occluding faces of the teeth also exposes normally protected dentin to abnormal conditions. Again, solution of the enamel by the process known as erosion exposes the dentin to external sources of irritation.

Pathology and Morbid Anatomy.—While it is true beyond question that the dentin is not, cannot be, the seat of inflammation, having no vascular system, its vital parts, the so-called dentinal fibrillæ, may give evidence of heightened irritability, and in all probability behave as any other formative tissue when subjected to stimulation and irritation, increasing the functional activity; giving rise, first, to hypersensitivity,

and then to constructive action, or the formation of dentin at the expense of the living portions, the tubuli becoming more attenuated. Certain it is that some molecular change must occur in the vital parts as the consequence of contact with a novel environment.

Symptoms.—The pain accompanying hypersensitivity of dentin is not localized to the point of affection, for the same reason that the sensations of the pulp, except that of throbbing, are rarely localized; the filaments do not possess the true tactile sense, a sense of location. The sensation of pain is elicited by contact of the dentinal filaments with acid substances; that is, acid substances are markedly irritating to the dentinal filaments; indeed, it is probable that this fact has much to do with the development of hypersensitivity of dentin, the acid of caries (lactic acid) subjecting these filaments to a constant source of irritation. The introduction of an acid substance into the mouth of a person who may have vital dentin exposed, is followed by a wave of gnawing pain, reflected usually along the course of contiguous nerve-filaments. The introduction of salt and very sweet substances is also followed by a similar though less marked reaction. The pressure of an instrument upon the dentin is attended by a flash of sharp pain, which continues for a time; but lessens if the contact be maintained. In this test the pain is localized in the affected tooth, the touch of the instrument being followed by a recognition of position by the tactile organ of the tooth, the pericementum. The response of such teeth to thermal applications is increased, the absence of the non-conducting layer of enamel permitting a more rapid reduction of dentinal temperature, so that the reaction of the pulp is correspondingly increased.

Diagnosis.—The only dental pain with which dental hypersensitivity may be confounded is pulp-pain proper. The diagnostic feature of pulp-pain is the reaction to changes of temperature. It is quite possible for marked pulp-irritation, followed by active hyperæmia of that organ, to occur without the presence of caries in a tooth. In diagnosing the condition under discussion the guide is the sensation of the patient, search being first made in the region described by the patient as the seat of pain. The first observation is directed to the finding of carious cavities; in the event of these being found, it is judged from the depth of the carious invasion whether there is a probability of pulp-disturbance. A differential test is made by contact of an instrument with the dentin, which, if hypersensitive, responds promptly. A drop of cool water is next dropped from a syringe into the cavity, when, if the pulp be in abnormal condition, there will be a sharp twinge of pain. In progressive caries this latter response is of increasing promptness and severity, until when the pulp is in a state of active hyperæmia or inflammation the application of a drop of cold water

is immediately followed by a paroxysm of throbbing pain. Observation is made to determine the presence of abraded surfaces exposing the dentin, and test by touch made. It is next noted whether points at the necks of the teeth respond promptly to the same test. Attention is directed to fissures and pits which may be present and expose the dentin.

Hyperæmia of the pulp, in the absence of caries, is indicated by a sharp paroxysm of pain produced by the application of cold to the enamel of the tooth.

Therapeutics.—The methods of treatment which have been followed for the relief of hypersensitivity of dentin, and the induction of such a degree of analgesia as will permit the necessary cutting of dentin, may be divided into general and local.

The general means of preventing pain may be placed under three heads: first, the administration of agents which lessen the function of the pain-perceptive centres of the brain—that is, abolish perception; secondly, the administration of remedies or the adoption of means to prevent the conveyance of painful impressions from the receptive end-organs to the pain-centres, or interference with transmission; thirdly, the prevention of the reception of abnormal stimuli.

The first group includes general anæsthetics and general anodynes. The inhalation of a few whiffs of chloroform or ethylic ether lessens the perception of pain. Chloroform is avoided in this connection on account of its dangers when used in the sitting position. Slight etherization, the inhalation being carried only to the benumbing-point, affords marked relief of the pain incidental to the cutting of hypersensitive dentin.

The administration of general anodynes, particularly the combination of morphia and atropia, has been found useful in this field:

℞. Morphiæ sulph., gr. $\frac{1}{8}$;
Atropiæ sulph., gr. $\frac{1}{150}$.
M. et ft. pil. No. 1.
S. To be taken one-half hour before operation.

The coal-tar derivatives, phenacetin, acetanilid, and others, are occasionally efficient. The preparations known as antikamnia (said to be a combination of acetanilid, caffein citrate, and sodium bicarbonate) and ammonol (acetanilid and ammonium carbonate, equal parts?) are to be preferred in this connection. The dose of the latter is gr. 10, administered one-half hour before operation.

The induction of the hypnotic state belongs in the category of means acting upon the nerve-centres.

Use of Alkalies.—Ever since a belief in the chemical nature of dental caries has been accepted, writers upon dental pathology have ascribed the hypersensitivity of dentin in caries to be due to the action of an acid, and have advised the use of alkalies to lessen the sensitivity. Those generally recommended are lime-water, prepared chalk, and sodium bicarbonate. These agents are commonly employed in cases of hypersensitive dentin at the necks of teeth; the powder is rubbed on and between the necks of the teeth before retiring at night. An effective agent of the same class is phénol sodique (sodium phenate). Used (diluted) as a frequent wash, it notably lessens the sensitivity of dentinal filaments. It is particularly useful for children.

Local Applications.—The usual method of treating the hypersensitivity is by the local application of analgesic agents. There are numerous remedies and agents which have been thus employed, and for convenience they may be grouped under the following headings:

1. Dryness and heat.
2. Cold.
3. Those which chemically destroy the protoplasm of the dentin.
4. Those which temporarily benumb and abolish the function of the receptive apparatus.

Heat and dryness are generally applied in conjunction, dryness being secured by means of blasts of hot air. Dentin, which protests against even the touch of an instrument while wet, has its sensitivity so lessened after the application of a rubber-dam and drying that it may be cut freely, in many cases without the aid of medicinal agents. So well is this recognized that isolation and drying of teeth are regarded as a necessary preliminary to cavity-preparation. The degree of insensitivity induced is in proportion to the dryness. The drying probably deprives the dentinal protoplasm of a portion of its water and inhibits the transmission of sensation.

Refrigeration, a well-known means of inducing local anæsthesia, finds application in this field. The temperature of the dentin of the tooth, which has been isolated by a rubber-dam, is reduced by sprays of highly volatile fluids; ether and chloroform, formerly employed, have been displaced by the more volatile substances, ethyl chlorid and methyl chlorid. These agents are contained in glass tubes having a minute orifice of exit; the cap of the vial being removed, the heat of the hand causes vaporization of the agent, which emerges as a fine but forcible spray. The full contact of the spray with the dentin should be made gradually to avoid painful response of the pulp.

Agents which chemically destroy the dentinal protoplasm form the most extensive group of dentinal obtundents. They include salts of metals, such as zinc chlorid and silver nitrate; carbolic acid and its

derivatives and like bodies; the cresols, etc.; mineral acids, notably sulfuric, chromic, and nitric; organic acids—trichloracetic and lactic acids (full strength), tannic acid; alkalies—sodium and potassium hydrates and carbonates.

Zinc chlorid, silver nitrate, and carbolic acid, all cause coagulation of the protoplasmic processes of the dentin. The mineral and organic acids chemically decompose both protoplasm and the calcified tissues. The concentrated alkalies mentioned chemically destroy protoplasm and bring about its quick dissolution. Like all active chemical substances, the extent of their action depends upon the freedom with which they are applied.

The caustic alkalies are used in connection with carbolic acid (Robinson's remedy):

℞. Potassium hydrate or
Sodium hydrate,
Acid. carbolic., } āā.—M.

Reduce the gelatinous mass formed with alcohol.

The application of any of these agents—metallic salts (coagulants), mineral or organic acids, and caustic alkalies—nearly always causes pain, the degree of suffering being usually in proportion to the depth of the cavity. The cessation of the pain is an indication of protoplasmic destruction. All of these agents are to be used in small amount and very concentrated; dilute solutions are ineffective. Carbolic acid and allied substances have an analgesic, instead of the primary irritating effect. They are paralyzants as well as coagulants, although less active than the other agents mentioned. Arsenic trioxid, arsenious acid, the agent used for the purpose of devitalizing the dental pulp, affects the protoplasm of the dentin profoundly, the effect being transmitted to the pulp, leading to the inevitable death of that organ.

The local analgesics proper include the essential oils and the sedative alkaloids; the oils of cinnamon, cloves, gaultheria, thyme. These exhibit the best effects in close proximity to the pulp. Thymol is the most powerful member of this group, and in addition is a strong antiseptic. The alkaloids which have been used are morphia, atropia, veratria, aconitia, and cocain. To be at all effective, these alkaloids must be used in concentrated form, so that the possible danger of poisoning by such powerfully toxic substances as aconitia, veratria, and atropia, contraindicates their general use; moreover, they are unnecessary. Morphia is ineffective. Cocain, the chief of all local anæsthetics, has but little effect upon hypersensitive dentinal filaments, although made into paste with glycerin it appears to be effective in some cases.

Absorption of the drug by the dentinal filaments does not occur, so that supplementary means are necessary to carry it into the filaments. This is accomplished through the agency of a galvanic current.

The tooth-pulp is excessively intolerant of changes of electric tension. If a tooth be perfectly isolated from its surroundings by means of a rubber-dam, and a very mild galvanic current be passed through the tooth, the positive electrode in the tooth-cavity, and the negative attached to the wrist or elsewhere, the pulp will respond promptly. If the current be continued, the response lessens, and finally ceases. Now, if the tension be increased by a considerable fraction of a volt, the pulp again protests; but if the increase be only a minute fraction of a volt, the pulp does not take cognizance of it; the voltage may thus, by imperceptible gradations, be raised from five volts to forty. The amount of electrical resistance offered by the tissues being extremely high, but a trifling ampèrage of current can pass through the tooth, even with forty volts pressure. If the dentin become dry, the resistance is much increased, as the only conducting-paths through the dentin are the dentinal tubuli when wet. Apparatus has been devised which permits the raising of current-tension by minute gradations, which has rendered available the cataphoretic use of medicaments in vital teeth.

Cataphoresis (Greek *kata*, down, and *phoreo*, I bear or bring) is, in technical parlance, the transference of substances from the anodal or positive pole of a battery toward the cathodal or negative pole. Cataphoresis, is to be distinguished from electrolysis, by which substances are decomposed and their elements carried from positive to negative, or from negative to positive poles, according to their polarity. In cataphoresis a substance is carried unchanged from the positive toward the negative pole after the manner of granules in protoplasm acted upon by the same force (see Chapter I.). If a tooth be insulated from its surroundings and a pellet of cotton moistened with a strong (10 per cent. to 24 per cent.) solution of cocain hydrochlorid and a platinum anode be placed against it, the cathode being attached to the wrist, the cocain will, by a current of gradually increasing tension, be carried along the protoplasm of the dentinal tubuli and induce local paralysis of the sensory function. If the action be continued with an increased voltage, the entire pulp becomes completely anæsthetic. The volume of current necessary to the cataphoresis of the cocain ranges from one-quarter to four milliampères.[1] The current-strength required ranges from five to thirty volts. Caution must be exercised to keep the cotton always wet, as dryness means greatly increased resistance and heat. The time necessary for the induction of dentinal anæsthesia ranges from about

[1] See *American Text-book of Operative Dentistry.*

eight to twenty minutes. J. A. Woodward [1] suggests the use of the galvanic current as a diagnostic means of determining the vitality of a pulp. A pulpless tooth does not respond when currents of comparatively high tension are passed through it.

It is customary to test the milder obtundents before resorting to cataphoresis.

Certain general rules should be observed in the treatment of hypersensitive dentin:

First. The most powerful agents, such as mineral acids, zinc chlorid, concentrated alkalies, etc., should never be used except in superficial cavities. With increase of cavity-depth milder agents may be substituted, until, when the pulp is nearly exposed, no more active agent than thymol should be employed.

Second. Resort should not be had to the active chemical agents until milder agents have proved inefficient. For example, zinc chlorid or sulfuric acid should never be the first agent used.

Third. All cavities should be isolated and dried before using obtunding agents.

Fourth. Arsenic should never be used unless destruction of the pulp is intended.

Fifth. None but new and perfectly sharpened instruments should be used to cut hypersensitive dentin, and the cutting should be accomplished with quick, light touches.

Prognosis of Caries.

The prognosis of dental caries is governed almost entirely by the thoroughness with which the indicated therapeusis is applied. In the absence of treatment, the disease, except in very rare cases, is continuously progressive until the greater portion of the tooth is disintegrated. Under proper and sufficient treatment the disease may be arrested at any stage of its progress, in any individual tooth; but the arrest of the process *in toto* can only be secured by a removal of its causes, both exciting and predisposing. If the exciting causes can be checked or removed, the predisposing causes will be in abeyance. Caries will persist or recur so long as the exciting causes of the disease are present.

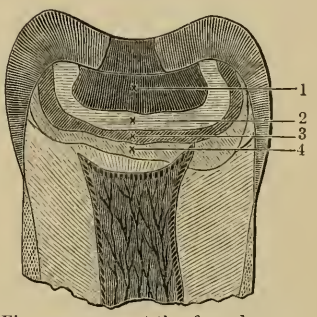
[1] *Proc. Academy of Stomatology*, Phila., 1896.

CHAPTER XVI.

DENTAL CARIES: THERAPEUTICS AND PROPHYLAXIS.

It is customary to divide the course of caries into a number of degrees or stages, each of which represents a more or less definite extent of invasion in the direction of the pulp, and in each of which the indicated therapeusis must be modified in accordance with the conditions. The first stage or grade, called superficial caries, represents the lesser degrees of softening and invasion of dentin, those cases in which as yet no evidences of any disturbance of the pulp have appeared. The second degree or stage is that in which pulp-protection forms a necessary part of the treatment. The third stage is that in which the depth of infection has endangered the functions or structure of the pulp. The fourth stage includes the cases of either actual invasion of the pulp by micro-organisms, or those which exhibit evidences of fatal structural changes in the vessels of the pulp.

Fig. 261.



Figures represent the four degrees of carious invasion.

The general therapeutic principle in the treatment of dental caries is the removal of all softened and infected tissue and the restoration of the original tooth-form by means of filling-materials. This general therapeutic principle is modified according to the depth of carious invasion—*i. e.*, the condition of the tissues involved.

Filling-materials as Therapeutic Agents.

Regarding as therapeutic agents all materials which by their physical or chemical properties affect the vital processes of tissues with which they may be brought into contact, it is evident that filling-materials themselves must be classed as therapeutic agents.

The theoretically perfect filling-material is one which possesses all of the physical properties of dentin and enamel, but is not, like these substances, soluble in lactic acid. The first essential of a permanent filling-material is, that it must hermetically seal a cavity in which it is placed, and undergo no physical or chemical change which may bring

about a failure of the hermetical sealing; that is, it must neither contract nor expand, and must be insoluble in organic acids, the solvents formed in the human mouth. It must be sufficiently hard and rigid to remain unaffected by the stress brought to bear upon it in mastication. It must be susceptible of a high polish, possess a harmonious color, and have a low rate of thermal conductivity. Not one of the filling-materials in present use possesses, in itself, all of these properties, although combinations of them, by adding together the individual virtues of single materials, remove several deficiencies of individual materials.

Gold, skilfully manipulated, may be made to hermetically seal a cavity. It remains chemically and physically unaltered in the conditions surrounding it; properly inserted, it withstands the stress of mastication, and is susceptible of a high polish, but its rate of conductivity is high, and its color is objectionable, although less so than that of amalgam.

Amalgams, as found commercially, expand or contract in setting or hardening, so that they do not permanently seal in their hardened state a cavity which they exactly filled while plastic. Under the stress of mastication amalgam flows, so that changes in the shape of a filling occur. They are insoluble in the organic acids of the mouth, but are acted upon by sulfur compounds and probably by oxygen, so that their color, primarily not so harmonious as that of gold, becomes more objectionable. They have a lower rate of conductivity than gold. By precise formulæ, properly treated[1] they may be made to remain stable as to contraction and expansion; but except copper be added to the alloy, the flow of amalgam-fillings cannot be entirely checked, in which event discolorations are more likely. Color may be improved by the addition of zinc and gold to a basal alloy of silver and tin. To insure stability of an amalgam-filling the primary alloy must contain not less than 65 per cent. of silver.[2] The value of all filling-materials depends primarily upon the skill and care with which they are manipulated.

It is still asserted by some homœopathic practitioners that amalgam-fillings, in consequence of the free mercury contained in them, are instrumental in perpetuating diseases of the buccal and pharyngeal mucous membrane, and that obscure general disturbances are also due to this cause. A hardened amalgam-filling contains no free mercury; all of this metal present is in chemical combination with the metals of the alloy. Metallic mercury is unaffected at ordinary temperatures by any chemical agencies found in the mouth, with the exception of sulfur

[1] See *American Text-book of Operative Dentistry.*

[2] Black, *Dental Cosmos*, 1896.

compounds; the statement that it is the "vapor of mercury" is absurd; mercury vaporizes only at a temperature of over 600° F.

Amalgam-fillings in contact with dentin in which putrefaction is in progress are frequently discolored by the formation of presumably black sulfid of silver and perhaps salts of mercury, the dentin being stained black.

Gutta-percha possesses the advantageous properties of entire non-conductivity, an agreeable color, comparative unchangeability in the fluids of the mouth, and hermetical sealing, but it is too soft to resist attrition and the stress of mastication.

Zinc oxychlorid in paste ($ZnO + ZnCl_2 + H_2O = 2ZnClHO$) is irritating to the dentinal filaments, the promptness of painful response being directly proportionate to the cavity-depth; it is antiseptic during setting and for some time subsequently; it is a non-conductor, is white, and when fully set is not sufficiently hard to bear the stress of mastication; its great drawback is its ready solubility in lactic acid. Moreover, it shrinks in hardening.

Zinc phosphate has an acceptable color; its rate of conductivity is higher than that of zinc oxychlorid; it does not contract in hardening, and is adhesive; but its hardness is not sufficient to permanently resist the stress of mastication, and it is also soluble in lactic acid, although less so than zinc oxychlorid. The properties of zinc-phosphate cements are governed by the chemical composition of particular specimens and in large part by their mode of manipulation.

Therapeutics of Superficial Caries.

The therapeutics of the first stage or superficial dental caries consists in the thorough removal of all softened tissue, including all enamel which has suffered from the action of lactic acid and all dentin which has become secondarily affected.

In cases of enamel caries, affecting the approximal surfaces of teeth, the practice has been advocated of filing away the affected enamel, when evident softening has not penetrated the entire thickness of the enamel, and polishing the cut surface, leaving, if possible, a space so shaped that the fluids of the mouth can wash freely through. Were it certain that all of the decalcified enamel could be removed by such means, and after operation a perfectly polished and properly shaped surface of unaffected enamel be left, no valid objection could be urged against the judicious following of the method; but, as Williams'[1] studies have shown, the advance of enamel caries is usually far in advance of the visible evidences of the decalcification; the practice of filing leaves practically unchanged the pathological conditions, and

[1] *Dental Cosmos*, 1897.

further progress of enamel decalcification at the original site is very likely to occur. In the light of the same discoveries, the prudent therapeusis is to excavate such spots of softening to the extreme margin of the softening. Whether or not the dentin is as yet affected, the excavation must be continued until sufficient dentin is invaded by the cutting-instruments to afford firm anchorage and support of the filling which is to restore the original contour. It will be observed that the physical and chemical properties of gold indicate it as the proper filling-material in these cases.

In similar cases at a later stage, when the dentin is invaded, the softened dentin is to be entirely removed, and with it all of the enamel overlying such dentin, until the cavity formed is bounded upon all sides by normal dentin and enamel. The removal of all softened dentin renders unnecessary the use of gemicides to the dentin, as softening is in advance of infection. In case of dentinal hypersensitivity in these cases it is usual to apply carbolic acid as the first obtunding agent; in which event, the same agent answers as a germicide also, but, as above noted, germicides are unnecessary in this stage, if excavation be thorough. In some cases, particularly those in which it is necessary to apply zinc chlorid, or even sulfuric acid, to obtund the hypersensitivity of the dentin, or cases in which the cataphoretic application of cocain must be made, the prepared dentin may become inordinately sensitive; moreover, the pulp responds with dangerous promptitude to changes of temperature. It is frequently advisable in these cases to treat the dentin as a wounded surface: procure surgical rest for a period; after touching the cavity-walls with one of the obtundent oils, insert a perfectly neutral and non-conducting filling-material—gutta-percha—for a period of some weeks. The alternative is to apply carbolic acid to the dentinal walls, dry perfectly, and coat the cavity-walls with a non-conducting varnish, such as that called Kristalline, or Cavitine, a solution of tri-nitro-cellulose in methyl alcohol. When evaporation has left a non-conducting film covering the cavity-walls a gold filling may be inserted.

A tooth in which thermal response is markedly increased through the presence of metallic fillings, is always to be regarded as one whose pulp is in danger of future disease and degeneration. The presence of a metallic filling in a tooth is almost certain to increase thermal response in some degree, but the extent and promptness should be reduced through the use of an intermediate non-conductor.

Superficial caries beginning in fissures or pits of the enamel is frequently of much greater extent than is evident from the orifice of the cavity. If the seat be a fissure, this should *always* be freely and broadly opened from one extremity to the other, and usually it is necessary to cut into communicating fissures. The excavating instruments should

follow every spot of softening until it is certain sound dentin has been reached in all directions, and, in addition, the enamel *must* be cut away until it is supported at all of the cavity-margins by healthy dentin. Cavities beginning in fissures are very deceptive as to their extent; a slight fissure may communicate with such a mass of softened dentin that the case belongs in the second class of cavities—deep-seated instead of superficial caries.

Therapeutics of the Second Stage of Caries.

In this stage of caries there is usually, although by no means always, an easily discoverable cavity of size. After the removal of ragged and overhanging enamel-margins, and of loose débris in the cavity, it is noted that the response to thermal impulse is painful and prompt. In washing such cavities, water at a temperature of about 100° F. should always be used; cold or very hot water being only employed in cavity-irrigation to test the promptitude of response upon the part of the pulp.

In treating hypersensitivity of dentin in such cases the strong agents zinc chlorid and the mineral acids are eschewed, the strongest agent admissible being carbolic acid, and even the use of this agent, acting as it does as a coagulant, is of doubtful propriety. A perfectly safe therapeusis does not admit of a stronger agent than an essential oil; for example, a saturated solution of thymol in glycerin or alcohol. It may be necessary to seal this agent in the cavity for twenty-four hours. Cocain cataphoresis is, however, regarded as admissible in all stages of caries.

The removal of the softened dentin in these cases forms a cavity of such magnitude that proximity to the pulp is evident. The softening has proceeded for a distance beneath the enamel, so that when all softened dentin is cut away from beneath the enamel the latter tissue overhangs, unsupported, the general cavity. These overhanging walls are cut away until the region of normal enamel is reached, and then it may be that the walls still overhang the general cavity. It is usually not necessary nor advisable to remove this portion of enamel. At the completion of excavation, the pulp is found to respond immediately to even a current of cool air, so that protection of that organ against thermal impulses is an imperative demand. It is in this connection that zinc phosphate finds its greatest and most useful field of application. It is used to replace the greater bulk of the lost dentin after the following manner: the dentinal walls are sterilized by an application of 25 per cent. pyrozone (25 per cent. solution of hydrogen dioxid in ether), are dried, and given a coating of non-conducting varnish. The varnish answers a double purpose; it lessens the moderate conductivity of the zinc phosphate and effectually prevents the action upon the dentinal walls

of any acid impurities in the cement. Many cement-fluids contain, as impurity, the acid sodium (dihydrogen sodium) phosphate, NaH_2PO_4. This, when present in a cement-filling, causes the persistent acid reaction noted in some cements; this is not only irritating to the dentinal filaments, but may cause superficial softening. Zinc phosphate, mixed stiff, is carefully packed against all of the cavity-walls until it is exactly flush with the enamel-margins and fills the cavity to near the dentin periphery; the cavity in the cement is given a retentive form to hold the veneer filling of metal which is afterward to be inserted.

Treatment of the Third Stage of Caries.

In the third stage of caries complaint is made that for some time the presence of cool or of hot fluids in the mouth is productive of a paroxysm of pain. After removing frail enamel-walls and the greater mass of softened dentin, including all of that underlying enamel, all of the decalcified peripheral dentin, it is found that further excavation would probably expose the pulp, which responds by painful paroxysm to even gentle currents of cool air. As a primary measure, even in the treatment of these conditions, irrespective of the filling operation, it is essential that the tooth and its neighbors be placed under a rubber-dam, where this device can be applied. This applies with equal force to operations for more extensive disease; the rubber-dam is used not only because it is impossible to effect perfect sterilization without it; but, in addition, it is only through the dryness and clear view obtained that therapeutic measures can be applied with that delicacy and precision necessary to their successful use.

The dentin-covering of the pulp is markedly softened, and the extent of bacterial invasion in the tubules is doubtful. The question arises, Shall excavation be made thorough, at the expense of probable pulp-exposure, or should it cease, and the softened dentin be permitted to remain? General clinical experience speaks for the latter course. The cavity in its present condition is washed out with a 5 per cent. solution of sodium dioxid, which neutralizes any acid present and is antiseptic.

It is evident that sterilization of the layer of dentin and pulp must be thoroughly effected to assure a probable success, for even under a perfect filling anaërobic organisms may make their way to the pulp and excite acute disease in that organ, which is already suffering from some degree of debility. Miller[1] demonstrated that the thorough sterilization of carious dentin requires much more time than is usually given to it. For example, to effect the complete sterilization of a very thin layer of dentin requires hours' *immersion* in strong carbolic acid. Bacteria were found in teeth which had lain in concentrated carbolic acid

[1] *Dental Cosmos*, 1891.

for nearly two hours. Watery solutions of antiseptics were found to penetrate the dentin more rapidly than essential oils; but the agents which effected prompt and complete sterilization, such as mercuric chlorid in 1–5 per cent. solution, zinc chlorid solutions, and others, are of questionable utility, owing to their probable deleterious action upon the pulp. It is preferable, therefore, to permit a harmless antiseptic to act for a longer period; for example, sealing a solution of thymol in the cavity over night. The application of a pledget of cotton moistened with a 25 per cent. solution of pyrozone promptly sterilizes very thin layers of dentin.

After sterilization and drying, a cavity-lining of zinc oxychlorid is indicated. Clinical records show that a layer of zinc oxychlorid placed over layers of decalcified or semi-decalcified dentin covering a pulp, are more frequently followed by favorable results than with any other material. In the light of present knowledge these benefits are attributed to the antiseptic property maintained by the material for some time. A paste of the cement is made, and a portion carried into the cavity, where it is immediately compressed against the cavity-walls by means of balls of bibulous paper which have been prepared for this purpose. All of the dentinal walls are covered to a depth of about one-sixteenth inch. As soon as the material has nearly set, usually in five to ten minutes, zinc phosphate is prepared and packed into the oxychlorid matrix, and brought flush with the cavity-margins. The surface-filling is then made of metal.

Treatment of the Fourth Stage of Caries.

The fourth stage of caries is that in which the pulp of the tooth is exposed before all of the decalcified dentin is removed. It is that stage of caries which includes in its treatment the operation of pulp-capping. A pulp which has been uncovered through the loss of overlying dentin and has been directly exposed to the fluids of the mouth, is almost certainly infected with micro-organisms; but as decalcification precedes bacterial invasion in caries, it is quite possible that the dentin covering a pulp may be decalcified before direct invasion of the pulp occurs. The pathologico-anatomical condition of the dental pulp is clinically judged by the symptomatology of the organ (see Chapter XVII.), and if the history of the case and the symptoms elicited at the time of examination give no evidence of disease of the organ, it is adjudged healthy, although the judgment formed may, in many cases, be no doubt erroneous. What is usually taken as presumptive evidence that a pulp has not undergone serious anatomical degeneration, acute or chronic, is that it has never been the seat of acute paroxysmal pain, and that it responds promptly to a current of

cold air. As will be shown in the chapter on Diseases of the Pulp, if paroxysms of pain have occurred, and if response to cool air is delayed, absent, or productive of prolonged pain, it is probable that serious anatomical degeneration has occurred in the pulp.

The details of cavity-preparation are the same as in the previous case, except that even the slightest pressure must be avoided in operating close to the pulp. That organ is very intolerant of even the slightest pressure. The free use of hydrogen dioxid in solutions of neutral reaction should replace other antiseptics. The use of strong caustic or coagulating antiseptics, even carbolic acid, is necessarily productive of injury to the pulp-tissue; the aim is to maintain the pulp in as nearly a normal condition as possible, not to establish an abnormal one.

Exposed Pulp.

An exposed pulp, even with the probability that it is non-infected and has suffered no serious anatomical alteration, is a condition requiring description and treatment in itself.

Diagnosis.—Clinically, pulp-exposure is divided into, first, cases in which the carious process has directly exposed the organ; secondly, those in which it is exposed through the removal of softened dentin covering it; thirdly, those in which it has been exposed by carelessness or by accident in excavating.

If the exposure be direct, it is detected visually. After isolation of the tooth, washing with tepid water, and drying, direct vision or a reflected image in the mouth-mirror may reveal the area of exposure as a round space occupied by a pinkish-red body. If the exposure be large, pulsation of the red body may usually be observed. The exposure may be so slight that it is invisible, the depth of the cavity, however, indicating that exposure probably exists. Truman[1] advises in these cases that finely carded cotton be gently passed over the cavity-walls, exposure being detected by the momentary pain produced when the fibres pass over the area of exposure. A burnisher passed gently over the walls of a cavity in cases of suspected pulp-exposure will usually elicit a quick start upon the part of the patient when the exposed spot is touched. The previous existence or presence of subjective symptoms, of course, is a diagnostic sign.

Prognosis.—Even presuming the absence of any history of active pulp-disturbance, the prognoses of the three classes of cases differ; prognosis is favorable in the reverse order of the conditions given. Again, the prognosis of exposed pulp is governed in great measure by the portion of pulp exposed; if a cornu of the organ be the site of exposure, the prognosis is more favorable than if the body of the organ be exposed. The

[1] *American System of Dentistry*, vol. i.

most favorable prognosis, all other conditions being alike, would associate with accidental exposure of a cornu of a pulp; the most unfavorable with direct exposure by carious invasion of the pulp, at or beyond, the neck of a tooth.

Treatment.—PULP-CAPPING.—It is more desirable that a pulp-capping should be absolutely neutral, than that it should possess active therapeutic properties. Theoretically it would be desirable to induce the pulp to form a plate of secondary dentin which should exactly repair the area of exposure; but as there is no means of exactly governing any stimulation which may be induced, it is preferable to use a substance entirely neutral therapeutically. A great number of substances have been recommended as pulp-cappers, but at present only two of them have general endorsement; these are a cement of zinc oxysulfate and a paste of one of the essential oils with zinc oxid. Solutions of gutta-percha in chloroform, disks of softened gutta-percha, and zinc-oxychlorid cement are also used, but not to the extent that the first mentioned are. Zinc oxychlorid, even when the fluid is dilute, may induce irritation, but in some cases it has proved an admirable material. Gutta-percha preparations, bland as they are, do not appear to serve so well as the cement of zinc oxysulfate or the zinc oxid and oil paste.

Whatever material be used, it is essential that not the slightest pressure be exercised in its application. The capping-paste is best and most accurately carried into place through the medium of a concave metal disk.

The oil and oxid paste is made by adding zinc oxid (cement powder) to a drop of oil of cloves until a moderately thick paste is made. The fluid of the oxysulfate cement is a saturated solution of zinc sulfate in water; the powder is chemically pure, zinc oxid, uncalcined. If the powder contain traces of arsenic, pulp-devitalization may ensue. Fluid and powder are made into a thin paste. The cement hardens very quickly into a white porous body of about the hardness of plaster of Paris. Whichever paste is used, the concave disk is filled with it, the disk being of a size to set firmly on the dentin all around the exposure. It is caught upon one side in the jaws of a delicate pair of pliers and carried quickly to the cavity; it is not set squarely over the exposure, but one edge is laid down first, and the disk delicately lowered until it rests upon the dentin and covers the exposure; the excess of cement oozes from the side of the disk last to touch the dentin. No sensation should be caused by the operation. If zinc oxysulfate has been used, the surplus paste is to remain; if the oil of cloves, the surplus is wiped away. The cap is retained in place and non-conductivity assured by lining the cavity with a layer of zinc phosphate, so thin that it will flow. It is the usual practice to complete the filling with zinc phos-

phate or gutta-percha, and if, at the end of six months, no evidences of disturbance have occurred and the pulp respond normally to applications of cold, a permanent filling is inserted.

Statistics regarding the success or failure of efforts at the conservation of the dental pulp have been presented in such manner as to make their value extremely doubtful. One reports the capping of all cases of exposure and gives the percentage of success. Another condemns the operation *in toto*, and states that exposure always indicates immediate devitalization of the pulp, and yet in both cases the probable morbid anatomical conditions which are deemed to warrant either course are not set forth. The clinical records of those who practise the operation after the method and under the conditions here set forth warrant the judicious practice of pulp-capping. No modern pathologist or well-informed practitioner ever questions the great advantage of having vital and normal pulps in teeth; pulpless teeth are less strong; they are liable to discolorations, and prone to pericementitis, even when skilfully treated.

Capped pulps may give almost immediate evidences of subjective disturbances, or the latter may not appear for years; but if the pulp remain quiescent for a year, a successful issue is accepted. Exaggerations or alterations in the mode of reaction to thermal impulses indicate an unfavorable issue, as do also pains referred to the region of the treated tooth.

Prophylaxis of Caries.

The prophylaxis or prevention of dental caries implies the removal of its exciting and, as far as possible, its predisposing causes. The removal of the exciting causes implies the destruction of ferments and the removal of fermentable material.

For the growth of the active causes of caries two conditions appear to be essential—that necessary for the life of all organisms, a food-supply, fermentable material; and, secondly, some undetermined bodily conditions which favor or deter their growth. The manner in which the constitutional condition reacts upon the secretions and tissues of the mouth, so that favorable or unfavorable conditions exist for the growth of lactic ferments, is purely conjectural; but it may be assumed that in a perfectly healthy individual, one having entirely normal oral conditions, the soil for the growth of organisms is most unsuitable.

It is freely conceded that bodily states react upon the oral tissues and alter their physiological relation, but the chief oral disturbances are, no doubt, of purely local origin. While it is unquestionably true that the correction of morbid conditions in other parts of the body may be followed by a cessation of oral disturbances, the extent of the changes thus

brought about are not comparable, in point of extent, with the changes induced by rational local therapeusis.

In general terms the prevention of dental caries begins with the correction of morbid physiology in all parts of the body. If general disease-causes—disorders of food-metabolism, of the blood-making organs, the blood-distributing apparatus, and the excretory organs—be in operation, a lowered vitality is present everywhere, including the mouth, and, no doubt, in the vital tissues of the teeth. Assuming that such causes and morbid conditions have been remedied, are irremediable, or not treated, the local measures are all-important. These are physiological, mechanical, and medicinal. The physiological and mechanical features both have to do with the removal of fermentable material. Normally the movements of mastication subject the teeth to friction, through the medium of food-stuffs; they cause an increased flow of saliva, which is pumped between the teeth and carried around and over them in currents. If the movements of mastication are lessened, not only is the extent of mechanical rubbing lessened, but the excretion of saliva is diminished, and after a time becomes altered in character. The coarser fibrous foods require more mastication and leave less débris than the soft, pulpy foods. Normal physiological use is, then, an important feature in the prevention of caries. If the individual can be persuaded to lessen the amount of cooked starches, pastry, etc., in the dietary, and substitute food requiring more mastication, the amount of fermentable débris is correspondingly reduced.

The presence of débris between the teeth and about their necks affords not only in itself the material for the generation of lactic acid, but furnishes a medium in which soluble sugars may be retained, which undergo transformation into lactic acid. The importance of freeing the teeth from food-deposits, inspissated mucus, calculi, etc., is generally recognized, it having been noted that caries is markedly lessened in well-kept dentures. The means of accomplishing this cleansing are largely mechanical—by the use of tooth-picks, brushes, and floss-silk. The tooth-pick and floss-silk are used to partially (they cannot completely) remove débris from the approximal surfaces of the teeth; the tooth-brush removes those from the occlusal, buccal, labial, and lingual surfaces. Were it possible through such means to thoroughly remove all débris, caries might be reduced to the position of an unusual disease; but it is because of their comparative inefficiency that painstaking cleansing, while lessening, does not prevent caries.

DENTIFRICES.

To aid the mechanical action of the tooth-brush it is usual to charge the brush with cleansing agents—dentifrices; these are in the form of

powders, pastes, and soaps. The powder should not be gritty; if it contain sharp particles, as charcoal, pumice, etc., the powder remains in the small spaces between the gums and teeth and acts as an irritant. The usual basis of all powders is calcium carbonate, in the form of precipitated chalk; this serves as a mild abrasive and neutralizes any free acids with which it is brought in contact. Magnesium carbonate or hydrate is to be preferred, as its particles are smoother and have less tendency to collect between the teeth. It is customary to combine with the chalk or magnesium from one-third to one-half its bulk of orris-powder for the supposed tonic influence of the latter upon the soft tissues. To this basal powder are added flavoring-substances and sugar, to render the dentifrice more agreeable. Oils of lemon, gaultheria, rose, etc., are used for flavoring, and sugar to sweeten the powder. Sugar, however, should be always omitted from dentifrices; it but adds to the fermentable material present in the mouth. To sweeten dentifrices saccharin should be used, of which but a minute portion is required; it is also an antiseptic.

Tooth-pastes contain about the same ingredients as an ordinary tooth-powder, made into a paste with honey and glycerin; their use is deprecated for this reason.

Tooth-soaps have the great advantage of saponifying and removing the fatty deposits from the surfaces of the teeth, large areas of retention which are but partially cleansed by powders. They are made by adding about one-third by volume of powdered castile soap to ordinary tooth-powder. Antiseptics may be combined with tooth-soap with marked advantage.

The last and a highly important consideration in the matter of prophylaxis is the destruction of the active organisms. The routine of tooth-cleansing includes the use of the tooth-pick, floss-silk, brush, and dentifrice, before the germicides proper are used. Unless the foreign deposits be first removed, the action of germicides is mechanically interfered with where they are most needed. It is essential that the antiseptic should be held in contact with the teeth for a long enough period to act as a germicide; to merely take it into the mouth and eject it in a few seconds accomplishes but little good. The most effective method of using oral germicides is that suggested by Ottolengui, to spray by means of an atomizer a solution of the germicide between all of the teeth and over all of their surfaces.

Miller[1] has tested the strength of solution of antiseptics admissible for this purpose, to determine which act most promptly and within the necessary time—about one minute:

[1] *Micro-organisms of the Human Mouth.*

Antiseptic.	Concentration.	Time necessary for devitalization.
Salicylic acid	1 : 100	$\frac{1}{4}$ minute.
Benzoic acid	1 : 100	$\frac{1}{4}$ "
Listerine	. . . . .	$\frac{1}{4}$–$\frac{1}{2}$ minute.
Salicylic acid	1 : 200	$\frac{1}{2}$ "
Bichlorid of mercury	1 : 2500	$\frac{1}{2}$–$\frac{3}{4}$ "
Benzoic acid	1 : 200	1–2 minutes.
Borobenzoic acid	1 : 175	1–2 "
Thymol	1 : 1500	2–4 "
Bichlorid of mercury	1 : 5000	2–5 "
Peroxid of hydrogen	10 per cent.	10–15 "
Carbolic acid	1 : 100	10–15 "
Oil of peppermint in agreeable strength	. . . . .	5–10 "
Permanganate of potassium	1 : 4000	More than 15 minutes.
Boric acid	1 : 50	" " 15 "
Oil of wintergreen	. . . . .	" " 15 "
Tincture of cinchona	1 : 18	" " 15 "
Lime-water	. . . . .	No action.

Combinations of antiseptics are desirable to act as stimulants and, at times, astringents to the soft tissues. Such prescriptions as listerine, thymozone, borolyptol, etc. (combinations of benzoic acid, thymol, formalin), and others, with such agents as eucalyptus, which exercise a favorable influence upon the vitality of the soft tissues, are used with much advantage. None but favorable results are noted after their long-continued use. It is customary to dilute them before using.

The times for the thorough cleansing of the teeth should be before retiring and after rising, particularly the latter. The periodical cleansing of the teeth by the dental operator is an important feature in caries prophylaxis.

SECTION IV.

DISEASES OF THE DENTAL PULP.

CHAPTER XVII.

CONSTRUCTIVE DISEASES.

Diseases of the dental pulp are both acute and chronic. According to the anatomical features, they may also be divided into constructive and destructive; and as to their character into functional and structural. The acute diseases are usually functional and destructive; in the chronic, structural and constructive changes are commonly noted. Constructive diseases of the dental pulp are those attended by the formation of deposits of new masses of dentinal substance. Destructive diseases are those which cause retrogressive and necrotic changes in the tissues of the pulp. The essential difference between the two classes of diseases is in the mode and character of the degeneration—the one is acute, the other chronic.

Pathologically there is no abrupt line of separation between those disorders usually termed diseases of the dental pulp and those which are described under the head of diseases of the live dentin. As soon as the dentin of the crown of a tooth is deprived of a portion of its normal protective covering, the enamel, either through chemical solution incident to the first phase of dental caries, or from mechanical abrasion, the vital portions of the dentin are subjected to new and abnormal conditions. These vital portions being in reality prolongations of the peripheral cells of the pulp, it is evident that the morbid conditions engendered by their exposure are expressions of pulp-disturbance, and we should expect to find reactionary effects upon the part of the pulp. Depending upon the severity of the irritation and the length or number of times sources of irritation have been in operation, evidences of functional and structural disorders in the body of the dental pulp are observed.

Post-mortem knowledge of structural diseases of the dental pulp is comparatively complete, but a parallel knowledge of the exact nature of the causes producing definite and recognizable conditions, together with the symptoms which precede and accompany the several morbid

states, is incomplete. In the absence of precise information as to the association between disease-causes, their symptoms and effects, physiological and pathological, the practitioner bases his diagnosis of the anatomical condition of the pulp on symptoms which he is enabled to elicit by certain tests, and by the history furnished by the patient. The tests applied and histories obtained, direct attention to the vascular system of the pulp as the primary cause of many, or most, of the conditions of the organ which are attended by paroxysmal and reflex pains. The reactions to tests occur both with and without exposure of the pulp to external sources of bacterial infection, although they are found in the vast majority of cases where bacterial invasion is a probability.

Symptomatology of the Pulp.

Writers upon dental pathology, during at least the past twenty-five years, have called attention to the fact that pain produced through the irritation of the dental pulp is rarely referred to its point of origin; that is, diseases of the pulp are, as a rule, characterized by reflected pains. G. V. Black[1] has clearly set forth the causes and reason of this phenomenon. "The pulp of a tooth is not its tactile organ; that is, it does not possess the sense of location. The only stimulus to which it responds in its normal state, when encased in an unbroken chamber of dentin, which is perfectly sheathed with enamel, is applications of heat or cold. Far removed in its normal state from situations in which a tactile sense could perform any physiological function, such a sense would be useless. Organs in which the tactile sense is absent and in which it would be perhaps superfluous, when the seat of disease have the pain incidental to the disease reflected to other parts; for example, in hip-joint disease, pain at the inner side of the knee is a diagnostic sign; in inflammations of the iris the pain is referred to the brow; pain at the orifice of the urethra is indicative of disease of the bladder, and so on. So with irritation of the dental pulp, the pain is indefinitely or vaguely located. In those cases where pain is referred to the tooth irritated, there are associated conditions which produce a response of the true tactile organ of the tooth, the pericementum."

The test by which the anatomical and physiological conditions of the pulp are judged is the specific stimulus to which the pulp is responsive—changes of thermal impulse. The phenomena induced by applications of water whose temperature is above or below that of the body, the extent and promptitude of the response, and the temperatures inducing it, are the only clinical means available for determining the condition of the pulp, the prognosis of its diseases, and directing the mode of their treatment.

[1] *American System of Dentistry*, vol. i.

It was pointed out by Black[1] that if a healthy tooth be isolated by a double layer of rubber-dam, and a jet of water at a temperature of 40° F. be directed against the tooth, a paroxysm of pain is produced. A jet of hot water will also induce a similar pain, and if the patient's eyes be shielded no difference in the sensations is noted. That is, the pulp responds to thermal stimuli, hot or cold indifferently. The organ is accustomed to variations of temperature between 60° and 105°–110° F., and within this range, in a condition of health, takes no apparent cognizance of this degree of change.

With a decrease in the amount of dentin covering the pulp—*i. e.*, with the advance of caries—the reaction to thermal stimuli increases in promptness, until, when the pulp is nearly exposed, the response is immediate. Succeeding this, is noted prompt response to lesser degrees of temperature-change, until the pulp comes to respond immediately to water at a temperature of 70° F., or thereabout, and slightly over the bodily temperature, 102° F. Later, another feature makes its appearance; instead of a sharp contraction-pain, applications of moderate thermal stimuli are followed by a heavy, throbbing pain. Later, similar pains occur in the absence of tangible external sources of irritation. In the ordinary sequence of events intense pain is later caused by hot applications, and cold applications afford relief.

The response to thermal stimuli may pursue the opposite course. The normally prompt response is followed by delays in reaction, until it is only after the continued application of cold to the exterior of a tooth that a paroxysm of pain is induced. In these cases there follows after a long time an increasing response to heat, as in the former instance, the reaction occurring only upon decided or prolonged heat-stimuli. Following upon the period of increased response to heat, in both cases there comes a period of quiescence, in which there is no response whatever to applications of intense cold, even that produced by the evaporation of a spray of ethyl or methyl chlorid—*i. e.*, the sensory function of the pulp is paralyzed.

These are the available subjective evidences of the anatomical condition of the pulp; while they indicate with a degree of accuracy, useful in clinical work, the alterations in the pulp, the exact relations between the reactions and the morbid anatomy of the organ are not entirely clear. In the light of present knowledge it is assumed that, in consequence of loss of the normal protective covering of the pulp, its sensory and perhaps vasomotor nerve-fibres become stimulated, overstimulated, irritated, then paralyzed by thermal stimuli in the progress of caries. The bloodvessels, which retained their tonus up to a certain point, suffer vasomotor irritation; next, paralysis leading to their dila-

[1] *American System of Dentistry*, vol. i.

tation and to the throbbing pain. Later, even change of posture is sufficient to cause distention of the paralyzed vessels, hence pain in resuming the reclining position. Stimulation by cold, until the later stages, causes a sharp continuous pain, ascribed to the paroxysmal contraction of the vessels; although unquestionably specific, sensory nerve reaction is involved. In the stages of paralysis, heat causes further distention of the vessels, and, if adventitious gases be present, causes their expansion with pressure upon nerve-filaments.

The decreasing and delayed response to thermal stimuli must be referred to two sources: first, an increase in the non-conducting covering of the pulp—*i. e.*, a lessening of the amount of the fluid contents of the dentinal tubuli and a thickening of the dentinal walls, which necessarily implies a recession of the pulp from its normal position; secondly, to degeneration of the sensory nerve-fibres themselves; and, thirdly, changes in the walls of or about the bloodvessels, which check vasomotor response and changes in the calibre of the vessels. These two classes of reactions still further emphasize the division of pulp-diseases into two types, the acute and chronic; the first class of reaction is associated with the acute destructive diseases, the second with the chronic constructive but degenerative conditions.

Constructive Diseases of the Dental Pulp.

The constructive diseases of the dental pulp include all the secondary dentin formations, tubular calcification, the formation of pulp-nodules, and calcareous degeneration of the pulp.

TUBULAR CALCIFICATION.

Definition.—By tubular calcification, or, to express the condition more accurately, tubular dentinification, are meant those changes that occur in the dentin which lead to an obliteration of the dentinal tubuli by constructive changes in the walls of the tubules.

Causes and Occurrence.—A mild degree of irritation, not passing the stage inducing constructive metamorphosis, and apparently caused by heightened thermal sensitivity. It occurs in the course of mechanical abrasion and erosion of the teeth, under metallic fillings; and probably a modification of the process precedes the slow invasion of dental caries. It occurs in some degree as a normal vital change due to age, and is common in persons who are victims of the gouty or rheumatic diathesis.

Effects.—The altered dentin becomes translucent, acquiring a horn-like appearance. While there is no doubt that the existence of this condition of the dentin delays the disintegration of the tissue, it does not prevent it.

SECONDARY DENTIN.

Formations of dentinal tissue in the pulp-chamber are of several varieties; the one under immediate discussion is that deposited upon and forming part of the pulp-chamber wall, lessening the volume of the cavity and of the dental pulp.

Causes.—A lessening of the volume of the pulp-chamber—*i. e.*, thickening of the dentinal walls—is a normal change of the teeth occurring with age. The change of senility is caused by a deposition of normal dentin; the lumen of the tubule lessens; the odontoblasts recede, and grow smaller and less in number. This change is accompanied by increase in the fibrous elements of the pulp; in other words, sclerotic changes occur with age.

It is to be remembered that the formative activity of the pulp is not exhausted until the pulp is almost obliterated, and when this occurs the organ has completed its physiological office and undergoes degeneration.

FIG. 262. FIG. 263.

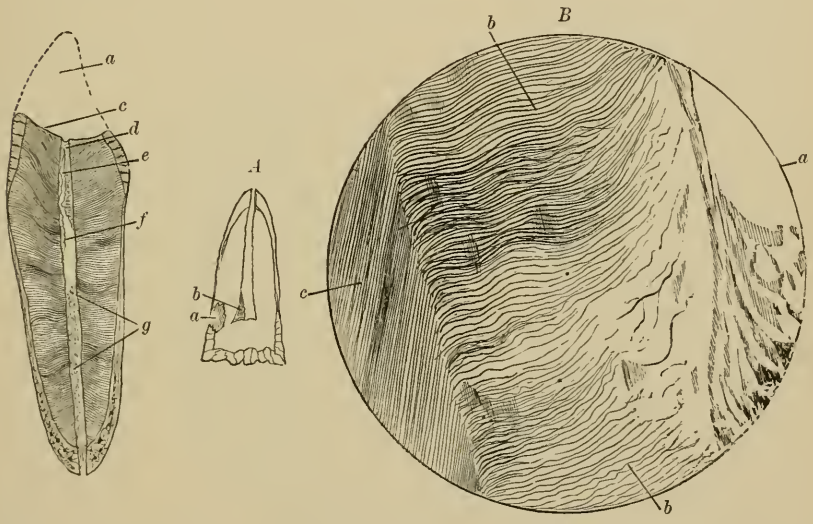


Fig. 262.—Secondary dentin, filling the pulp-chamber in case of abrasion of a cuspid tooth: *a*, portion lost by abrasion; *c*, abraded surface; *d*, secondary dentin, filling a portion of the pulp-chamber, and acting as a protection to the pulp; *e*, slender point of the pulp; irregular deposits are seen on the walls of the pulp-chamber, as at *f*; *g*, cylindrical calcifications in the root-portion of the pulp-chamber.

Fig. 263.—Calcification, or deposit of secondary dentin, resulting from caries of an incisor: *A*, diagram of section of incisor, showing caries at *a*, and secondary dentin at *b*. *B*, illustration magnified 200 diameters, to show the tissue of the secondary dentin: *a*, pulp-chamber; *b*, *b*, secondary dentin; *c*, primary dentin. It will be noticed that the dentinal tubes in the secondary dentin gradually disappear, giving place to a clear calcification. (Black.)

As will be seen in the discussion of the several constructive changes, variations in the conditions of the teeth may bring about the premature

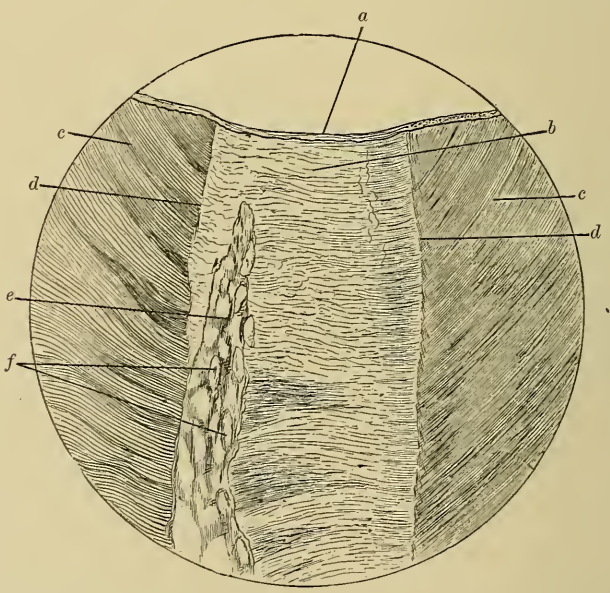
exhaustion of the formative activity, with its consequence, atrophy of the pulp.

The formation of secondary dentin implies the action of a localized stimulation of formative activity of the odontoblasts. It is probable that all of the cases of localized irritation may by careful investigation be resolved into an increased conductivity; that is, a lessened non-conductivity through a defined path.

Secondary deposits are commonly found associated with abrasion, erosion, the slow advance of dental caries, and with metallic fillings in proximity with the pulp.

Pathology and Morbid Anatomy.—The formation is noted opposite some area of injury (Figs. 262, 263), and may be easily distinguished from the normal dentin. Viewed macroscopically it is usually seen to differ from normal dentin in its degree of translucency (Fig. 264), and viewed microscopically there is a sharp change of direction

FIG. 264.



Secondary dentin, from the same specimen as Fig. 262, magnified sufficiently to show the difference in primary and secondary tissue: *a*, abraded surface of crown: *b*, secondary dentin; *c*, primary dentin; *d*, junction of primary with secondary dentin; *e*, remains of pulp-tissue; *f*, small oval masses of calcific material. (Black.)

of the tubules which abruptly marks off the secondary from the normal dentin. So long as the stimulation is uniform and localized, the new formation appears to be limited to an amount which will equalize the rate of conductivity in the dentin.

The growth excited by caries presents some features differing markedly from those excited by abrasion.[1]

Growths in Abrasion.—The growths excited by abrasion have more regularity of structure, and the gradual obliteration of the pulp-chamber occurs in more regular lines (Fig. 265). In cases affecting double or triple rooted teeth Black found the deposits limited to the pulp-chamber and to the bulbous portions of the pulp, the diameter of the root-canal being diminished only at its entrance; the deposits may extend for some distance up the canals, but never far, a condition different from that noted in deposits excited by other causes.

FIG. 265.

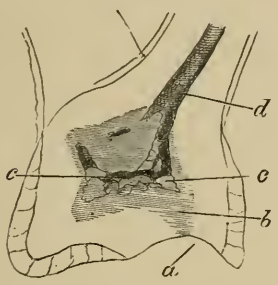
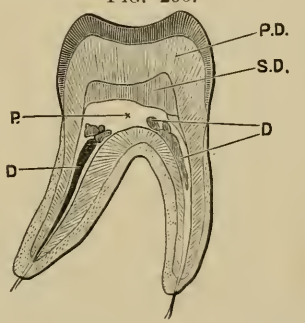


Illustration of the narrowing of the pulp-chamber in a molar (superior) by the deposit of secondary dentin resulting from abrasion, showing the portions of the chamber in which the deposit usually occurs. The light-shaded portion (*b*) shows the original dimensions of the chamber, which in this instance seem to have been pretty large; *a*, a point of deep abrasion; *c*, *c*, remaining pulp-chamber, which is mostly filled with irregular masses; *d*, one of the root-canals. It will be observed that the narrowing of the root-canal is within the original pulp-chamber. (Black.)

"Secondary growths in cases of abrasion are not confined alone to the abraded teeth, but other teeth which have escaped wear may be affected in equal degree. In all of these cases there is direct evidence that the odontoblastic layer has been stimulated to increased activity and produced the regular secondary deposition."

FIG. 266.



P.D., primary dentin; *S.D.*, secondary dentin, *P*, pulp-chamber; *D*, nodules.

Growths excited by caries usually lack the regularity observed in the former cases, the difference being, no doubt, attributable to the nature and intensity of the irritation produced. While, as shown, stimulation of the dental pulp results in a somewhat regular and outlined functional activity, other grades of pulp-disturbance lack this continuous degree of response, and formations are irregular; and by the same rule the existence of irregular deposits is an indication of grades and varieties of pulp-disturbances in excess of the stimulation; they indicate irritations and hyperæmias. This is well illustrated in the case from which the appended figure (Fig. 266) was taken. The carious

[1] Black, *American System of Dentistry*, vol. i.

process was progressing slowly in the anterior segment of an upper first molar, the second bicuspid being absent. When the cavity had reached the second stage of invasion, it was filled with zinc phosphate. At this time it responded normally to the thermal test. After four years, vague discomfort was felt in the region, and in another year was referred to the filled tooth, which responded faintly to hot applications and not at all to cold. Diagnosis: pulp in last stages of degeneration. Upon opening the tooth the portion of the chamber opposite the original caries, and beyond, was found much contracted by a deposit of secondary dentin; the bulbous portion of the chamber and canals contained large loose dentinal tumors, that in the palatal root almost filling the canal. These deposits were of conglomerate nature, the elements of pulp-tissue being caught in their substance.

The formation of regular deposits of secondary dentin, causing uniform or nearly uniform contraction of the pulp-chamber, has no clinical significance in point of therapeutical indications. The process must be regarded as conservative in character, although unquestionably the formation of secondary dentin hastens the physiological exhaustion of the pulp-tissue. Black[1] calls specific attention to the changes in the character of extensive secondary deposits, that at first the new structure contains nearly the normal number of tubules, which later become fewer —*i. e.*, the odontoblasts have lessened in number and later structureless granular dentinal matter is found.

Other forms of calcic deposits are found in the pulp-chamber, mechanically displacing portions of the pulp-substance, occupying the interstices, enveloping the pulp-elements; and again the tissue-elements themselves appear to undergo calcareous degeneration. These all appear to be due to the occurrence of higher grades of pulp-disturbance and vascular reaction than represented in the case of orderly deposits of true dentinal substance. They probably arise in consequence of repeated hyperæmia of low grades, of venous hyperæmia, and of those conditions indefinitely known as chronic inflammations. Their association with these conditions, however, is not clear enough to definitely classify them as the consequences of any determined pathological condition of the pulp. While they are unquestionably evidences of pulp-degeneration, and should be so classified, they are, for the sake of convenience, grouped under the head of constructive changes. The fact that they are degenerations is to be constantly borne in mind.

PULP-NODULES.

Definition.—Pulp-nodules (pulp-stones, nodular calcifications) is the name applied to defined masses of calcic material occupying portions

[1] *American System of Dentistry*, vol. i.

of the pulp-chamber and causing displacement of the pulp-substance.

Occurrence.—While these growths may occupy the pulp-chambers of teeth in which the pulp has been the seat of direct irritation, their occurrence is by no means confined to such teeth. They are found, not only in teeth which have suffered abrasion, erosion, and slowly progressing caries, but, as pointed out by Black, they may, and frequently do, form in other teeth of the same denture which are not directly involved in the irritation. This investigator notes that irritation of the pulp of one tooth of a denture very frequently causes a general hyperæsthesia of the pulps of all of the teeth. This is particularly notable in the type of persons classed as neuralgic. It is also common in persons of the gouty diathesis. It should be remarked that a general pulp-hyperæsthesia is frequently the precursor of an acute outbreak of gout in such persons. Nodules are found much more frequently in the teeth of middle-aged persons than in those of youth, although they may be present as early as the fifteenth year. They occur more frequently multiple than single. Some of the larger nodules are evidently formed by the coalescence of smaller ones.

Pathology and Morbid Anatomy.—The structure of pulp-nodules does not resemble that of dentin; they possess about the same degree

FIG. 267.

Section of a pulp-nodule, showing many calco-spherites, as pointed out by *a*, *a*. (Black.)

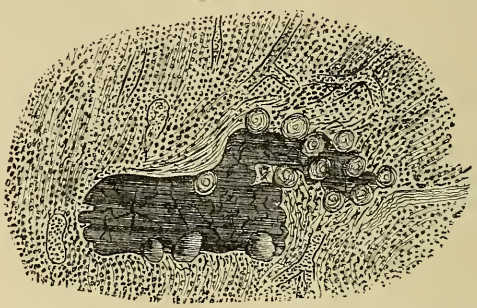
of translucency and hardness. Outwardly they may assume almost any form; they range in size from minute bodies to a size sufficient to almost obliterate the pulp (Fig. 265).

A section of a nodule exhibits the presence of a number of concentrically laminated bodies, recognizable as hardened calco-spherites. Black found them "to rarely make up any considerable portion of the

bulk of the nodule. The remainder of the nodule is made up of structureless dentinal matter.

He also found deposits in the pulp which throw light upon the possible origin of nodules in some cases, and to some extent upon the conditions under which they may be formed (Fig. 268). In the pulp of a second

FIG. 268.



Deposit of calco-globulin within the tissues of an inflamed pulp. (Black.)

molar of a girl aged fifteen, in which there had been decided subjective evidences of pulpitis recurring at intervals, for a period of two months, he found a mass representing a pulp-nodule in its soft state. "About one-half of the coronal portion of the pulp was involved in the inflammation; lying a little inside of the layer of odontoblasts were several masses similar to Fig. 268, having globular forms in their mass or attached to their margins. The globular bodies present the laminated appearance of calco-spherites." These masses may in all probability be interpreted as intermediate products in the formation of nodules; they have not yet become calcified. The conditions of calcification in enamel and dentin are not definitely known (Chapter VII.), so that the mode of calcification in pulp-nodules is also unknown. Black suggests, following the experiments of Rainey, Ord, and Harting, that the chemical conditions for the formation of calco-spherites appear to be a solution of albumin, calcium salts, and an excess of carbon dioxid; these conditions are realized when there is an excess of venous blood in semi-stagnation, the conditions which exist in varicose veins, a pathological state in which concretions (phleboliths) appear in the veins. "When a venous thrombus is but partially replaced by connective tissue the remainder of it may become calcified, forming phleboliths.[1] While this may and no doubt does serve to explain calcareous degenerations, it fails to explain the formation of pulp-nodules, which exist in teeth which have never been the seat of caries, and whose pulps exhibit no structural change other than the presence of nodules. Moreover, the presence of hardened calco-spherites, and even an amorphous

[1] Ziegler.

calcified, translucent mass, implies secretion rather than chemical precipitation. They are, however, not to be classed as secondary dentin, because they lack the distinguishing feature of dentin—dentinal tubules.

Symptoms.—Multiple nodules may exist in a dental pulp and give rise to no evident symptoms whatever, as is shown by their presence in extracted teeth, many of them free from caries, and in which there was no history of pain. On the contrary, the pulp of a tooth may be the seat of intractable pain without a depth of carious invasion which would lead to the inference of acute pulp-disease; and relief only be secured through devitalization of the pulp, which upon examination may reveal a small pulp-nodule.

The symptoms attendant upon the presence of nodules, so far as they can be made out, appear to be of two types—those associated with small and those with extensive deposits. Reflex pain is the common associate of both.

Small Deposits.—While it is true that pulp-nodules exist in apparently sound teeth without inducing pain, yet the pulps of teeth containing them become excessively hyperæsthetic under what are ordinarily mild sources of irritation. This is manifested, first, through the contents of the dentinal tubuli; the dentin becomes exquisitely sensitive, and cool water directed into a shallow cavity produces a paroxysmal and excruciatingly painful response from the pulp. In the absence of direct, extraneous irritation of the pulp, the dental symptoms may be absent, but a persistent neuralgia may be located at some distant point. Pain *in* the ear is a frequent symptom. Occasionally an obstinate scalp neuralgia, with the existence of a hyperæsthetic spot, appears. Pain in the eye, with tenderness over the supra-orbital foramen, is also common. The pain may be recurrent or persistent. If, in the absence of a more probable explanation of the pain, a pulp-nodule be suspected, and arsenical applications be made to devitalize the pulp, it is found that not only is intense pain caused, but examination after from forty-eight to seventy-two hours shows the pulp to be still vital and hypersensitive; and in order to effect its destruction repeated applications and large doses of arsenic must be used.

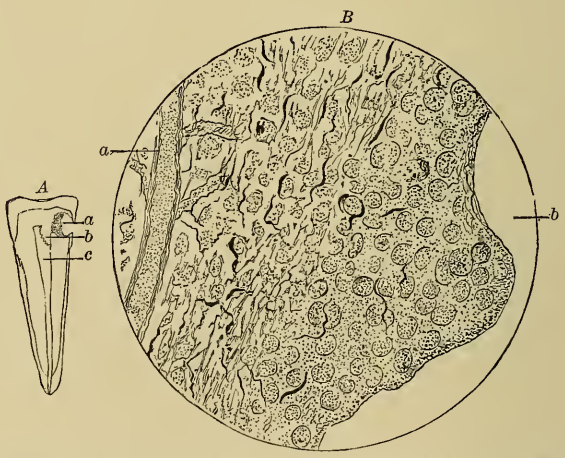
Large Deposits.—In extensive deposits of pulp-nodules the dentin may be almost devoid of sensation, and applications of heat or cold, even in large cavities, may be followed by delayed and faint pulp-response. Such cases, however, commonly give a history of reflex neuralgia and vague dental pains extending over a period, it may be, of years. Their diagnosis may only be made after devitalization of the pulp and the finding of the nodules in the pulp-chamber or pulp-substance. The tardy action of arsenic is also observed in these cases, it being frequently necessary to devitalize the pulp piecemeal.

Treatment.—When pulp-nodules have been diagnosed as the probable source of dental pain or of reflex neuralgia, the therapeutic indication is the devitalization and removal of the pulp. The first arsenical application should contain a much greater amount of cocain than arsenic. In forty-eight hours a stronger paste may be applied. In the more obstinate cases, it may be one or two weeks before devitalization is complete. Uncomfortable symptoms referred to the teeth may persist for some time subsequent to devitalization and thorough removal of the pulp. Evidences of pericemental disturbance, tenderness upon percussion, may appear. The uncomfortable symptoms disappear, as a rule, if a saturated solution of menthol in chloroform be pumped in the canals, the cavity sealed, and the gum at a distance from the teeth be painted with tincture of iodin.

CALCIFIC DEGENERATIONS OF THE PULP.

Calcific degenerations of the pulp are of two types: one occurs as a sequel of the degenerative changes of age—atheroma; the other, as calcic deposits in tissues of the pulp which have been the seat of acute or subacute vascular derangements. Calcareous degeneration, as

FIG. 269.



A, diagram of a section of a central incisor, with a proximal decay at *a* which seems to have penetrated the original pulp-chamber, but the opening is closed by calcification at *b*; *c* marks the position of a detached mass of calcific material that was lost in mounting the section. *B*, shows the appearance of the calcific deposit; this seems to be a calcification of inflamed or cicatricial tissue; at *a* there is the appearance of a bloodvessel; *b*, pulp. (Black.)

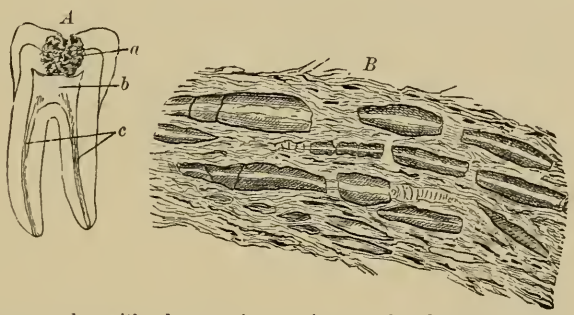
pointed out in Chapter IV., has as a precursor other degenerative changes; it is, in fact, a deposition of calcic material, calcium and magnesium phosphate and calcium carbonate, in already degenerated tissues.

Causes.—The causes of calcareous degenerations of the pulp are therefore in previous diseases of the pulp which have induced degen-

erative changes in the pulp-tissue. Prominent among these Black found grades of pulp-inflammation, of hyperæmia (Fig. 269).

Pathology and Morbid Anatomy.—The calcic material, unlike the cases of nodular calcification, incloses the anatomical elements of a pulp in process of degeneration in a mass produced by deposition, not secretion. In the root-portions of pulps in which fibrous elements have become pronounced the calcification may be tubular or cylindrical in character, the nature of the calcareous masses being apparently a deposition about and along the fibres (Fig. 270).

FIG. 270.



A, outline of a lower molar, with a large carious cavity at *a*; *b*, pulp-chamber; the shaded portion, *c*, was occupied by cylindrical calcifications. *B*, cylindrical calcifications. (× 100.) (Black.)

OSTEODENTIN.

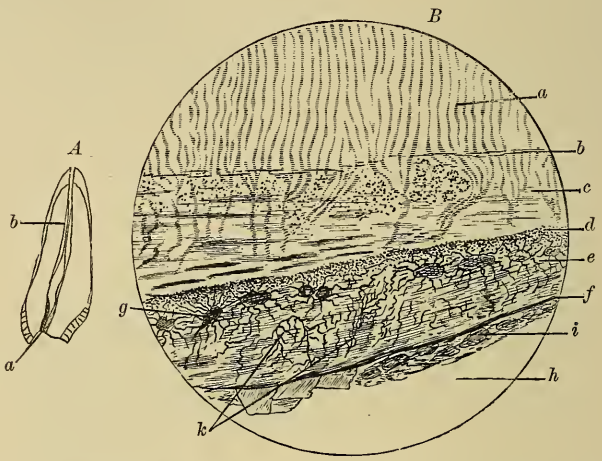
Tomes[1] states that secondary dentinal deposits may assume the character of osteodentin, a form of dentin found in the teeth of some animals, in which the tissue presents combined characters of both bone and dentin, citing the example also that elephant tusks are frequently repaired with osteodentin after injury. The specimen illustrated (Fig. 271) was taken from a case in which the coronal portion of the pulp-chamber was almost obliterated by a deposit of secondary dentin. It is difficult to conceive the origin of the osteoblasts in this case; the evidences of former odontoblasts are plain; but whether odontoblasts under altered conditions have become osteoblasts is a matter of conjecture.

To recapitulate, there appear to be at least three distinct types of calcic formations in the pulp-chamber. First, secondary dentin, in which the odontoblasts recede from their original positions in consequence of a new formation of dentinal material, leaving behind them processes which give the new formation the character of dentin. The process, in the main, is associated with a mild and continued irritation leading to a continued hypernutrition. After a period the physiological activity of the odontoblasts ceases and they undergo atrophy. The

[1] *Dental Anatomy.*

second class of cases, nodular deposits, possess the physical and chemical properties of dentin without its anatomical characteristics; it is dentinal substance, but not dentin histologically; hence it differs from dentin in not being formed through the agency of odontoblasts; it is

Fig. 271.



Osteodentin: *A*, outline of incisor, showing a narrowing of the root-canal at *b* by a deposit of osteodentin. *B*, illustration of the tissue: *a*, primary dentin; *b*, line of the beginning of a growth of secondary dentin; *c*, secondary dentin; *d*, layer of granular matter; *e*, osteodentin; this has the lacunæ at *g* and dentinal tubes at *k*; *f* seems to be the surface of the osseous deposit; *i*, irregular crystalline deposits; *h*, the pulp-chamber. × 350. (Black.)

the product of secretory instead of formative action. Whether nodular deposits are first formed as a soft mass, and subsequently calcified, is not known. They appear in general to be the result of marked and irregular irritation of the pulp. The third form are calcareous depositions about anatomically degenerated tissue occurring as a secondary process to degeneration and always indicating the near death of the pulp.

CHAPTER XVIII.

DESTRUCTIVE DISEASES OF THE DENTAL PULP.

The next class of pulp-diseases are those of an acute character, although chronic diseases may arise as sequelæ of the original conditions. They are essentially destructive in character and attended by prompt degeneration of the pulp-tissues. The most important clinically are those having an evident association with disorders of the bloodvessels of the pulp.

Hyperæmia of the Pulp.

Hyperæmia of the pulp is an excess of blood in the more or less dilated vessels of that organ. It is of two forms, active or arterial hyperæmia, and venous or passive hyperæmia or congestion. These two classes differ in their probable direct causations and in effects.

ACTIVE HYPERÆMIA OF THE PULP.

Definition.—Active or arterial hyperæmia of the pulp is an excess of blood in the dilated arteries and capillaries of the pulp.

Causes.—The most common cause of active hyperæmia of the pulp is a lessening of the non-conducting covering of the organ, enamel and dentin, leading to an increased response and continued irritation of the pulp through thermal stimuli. A similar condition consists in the presence of large metallic fillings in close proximity to the pulp, through which abnormal thermal stimuli are received. Fillings through which prompt pulp-response to thermal changes are felt are a direct menace to the continued health of the pulp. "The vigorous use of sandpaper disks in finishing large fillings may and does precipitate an attack of pulp hyperæmia."[1] The loss of tooth-substance mentioned may be either through abrasion, erosion, or caries. The condition frequently occurs without direct exposure of the dental pulp.

Symptoms.—When the pulp has lost much of its protective covering, its response to thermal change becomes increased. So long as a quick, sharp pain is produced by contact with cold or hot substances, ceasing immediately, and only reappearing in response to direct stimuli, no serious vascular disturbance is inferred; but when paroxysms of sharp pain lasting from many minutes to hours follow upon an application of cold to a carious cavity, an unbroken enamel-surface, a filling,

[1] Black.

or an area of erosion or abrasion, a disturbance of the vessels of the pulp is suspected. The pains, in the absence of direct and intentional irritation, are, as a rule, but vaguely located. It is common to have the pain referred to somewhere in the region of the affected tooth; rarely to the tooth itself. It is not at all unusual to have the patient refer the pain to an entirely sound tooth at a distance from the one affected; a tooth in the opposite jaw may be declared to be the seat of pain, and it may require the application of the thermal test to the offending pulp to convince the patient of the error of location.

For some period previous to, and it may be after, an attack of acute paroxysmal pain, trigeminal neuralgia of the side may be complained of. A favorite location of this pain is in the ear. As a rule, when an upper tooth is affected the pain is located in the superior maxillary division of the fifth nerve; if a lower, to the inferior maxillary division. The pain varies in intensity from a vague uneasiness to an acute neuralgic attack, with tender spots over the emergence of the nerve-tracks, at the supra- and infra-orbital, and mental foramina. The neuralgic pains are not always constant; they may disappear from the second or third division of the fifth nerve and appear in the first.

The proof of the direct connection between the pulp-pain and the neuralgia may in some cases be clearly made out by the thermal test. When a jet of cool water is directed against the tooth whose pulp is affected, it may produce, in addition to a local pain, an aggravation of the neuralgic pains.

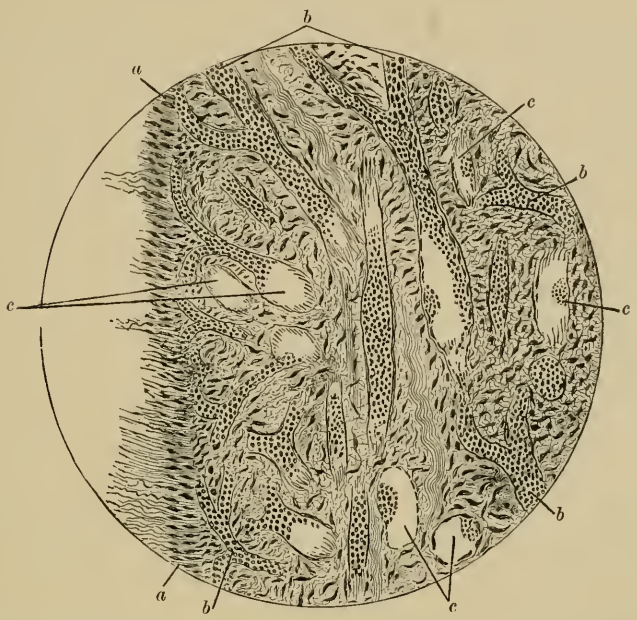
Pathology and Morbid Anatomy.—The one distinctive and characteristic anatomical condition associated with active hyperæmia is an irregular dilatation of the vessels of the pulp.[1] Fig. 272 represents a section of the pulp of a tooth extracted during a paroxysm of acute pain—"acute paroxysms of pain lasting for an hour or more were occasionally occurring in consequence of very trivial changes of temperature; the condition had existed for several weeks." In some cases of a similar character—*i. e.*, presenting the same symptoms, but extracted during an interval of quiet—nothing remarkable is presented; the veins of the bulb may be abnormally large and contain more blood than usual, while the arteries will be almost or quite empty and the injection of the capillary system wanting; that is, the affected arteries have recovered their calibre, if not their tone. Black found the varicose enlargement of vessels so common (Fig. 273) as to be a characteristic. Salter[2] first called attention to the dilatation of veins into ampullæ, describing them in connection with ulceration of the pulp, as due to engorgement and overtension of the veins.

The most rational explanation of the dilatation of the vessels is that

[1] Black, *American System of Dentistry.* [2] *Dental Pathology and Surgery.*

it is an irregular paralysis of vessel-walls—*i. e.*, of vasomotor nerves. Whether the more usual painful responses of the pulp to thermal stim-

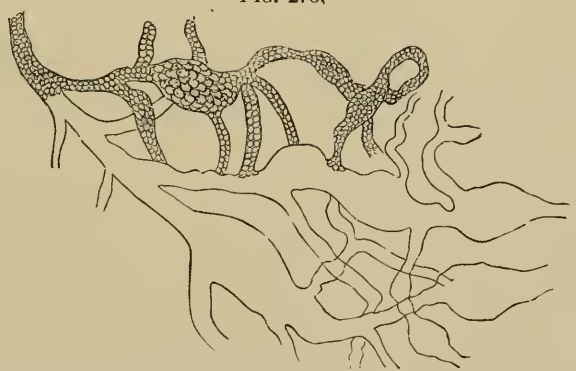
FIG. 272.



Hyperæmia of the dental pulp, showing the natural injection of the vessels: *a*, *a*, membrana eboris, or layer of odonoblasts; *b*, *b*, *b*, *b*, vessels distended with blood; *c*, *c*, *c*, *c*, points from which the blood has fallen in handling the section. (Black.)

uli are due to the stimulation of vasodilator fibres, which causes a transitory hyperaemia, is a matter of doubt; but the pathologieal eonditions

FIG. 273.



Dilated bloodvessels from the dental pulp in hyperæmia, from tooth extracted during a paroxysm of intense pain. (Black.)

noted in pronounced hyperæmia signify a paralysis of vasoconstrictor fibres. Subjected to repeated over-stimulation, they become inactive

and the vessel-walls yield to the pressure of the blood-column. Black's researches indicate that the vessel-walls may recover their tone and the vasoconstrictor nerves their functional activity after paralysis.

Diagnosis and Prognosis.—Diagnosis of hyperæmia of the pulp is made through observance of a combination of signs and symptoms. The symptoms leading to its detection are paroxysms of pain induced by thermal stimuli, and a history of pain in the region in which this response is elicited. The signs of the condition in the order of their importance and frequency are carious cavities, the presence of large metallic fillings, deep erosions or abrasions, and, again, fractured enamel exposing the dentin, or metallic crowns on teeth containing vital pulps.

As a rule, the case presents a history of paroxysmal pain for a period; either a single or several attacks. While, usually, pulp hyperæmia is only associated with deep cavities of decay, it is occasionally found as an accompaniment of limited and comparatively superficial dentin exposures. The water used in testing the pulp-reaction should not be at a lower temperature than 60° F., and then be applied only in drops, never in a forcible stream. A normal pulp will rarely respond painfully to a few drops of water at the temperature named, flowed into a cavity; but a hyperæmic pulp will almost invariably respond vigorously. As a rule, a current of air from a chip blower is a test of sufficient severity.

In the absence of a carious cavity the source of the pain is to be sought in large fillings, testing each tooth by dropping cool water on the filling; in cases of erosion or abrasion the test is made upon the exposed dentin. The tooth which responds in a quick paroxysm of intense pain, passing away slowly, is diagnosed as the seat of pulp hyperæmia.

The prognosis of this condition is important as determining the course of treatment. Governed by clinical experience, many operators invariably devitalize and remove a pulp which has been the seat of more than one attack of paroxysmal pain. Others attempt the conservative treatment of the organ even when it has been judged from the symptoms the seat of repeated hyperæmia, and report that success usually attends the effort, provided due precautions have been taken as to antisepsis, the character of pulp-cupping, and to non-conductivity of the pulp-covering.

Black[1] has shown the capability of the pulp to recover from repeated hyperæmia; that is, as regards the condition of its bloodvessels; and the records of observers (notably Louis Jack[2]) show that after years

[1] *American System of Dentistry*, vol. i.

[2] *American Text-book of Operative Dentistry.*

the pulp responds normally to thermal stimuli, proving its continued vitality, aye more, its health. It is possible that those who condemn the attempts at conservation of the pulp after hyperæmia have confounded this vascular condition with serious degenerative changes. Properly treated, the prognosis of active hyperæmia of the pulp is, upon the whole, favorable.

Treatment.—The therapeutic principles involved in the treatment of this condition are : the removal of the source of irritation and the securing of physiological rest ; the latter can only be secured through the removal of the former. The treatment is directed toward immediate relief of the existing condition and the prevention of its re-occurrence. If a carious cavity exist, it is to be freed from debris ; and the grosser portions of the carious dentin are removed ; the pulp, if unexposed, is to have the layer of softened dentin covering it left unremoved. Sedative agents are imperatively called for, that most commonly employed being carbolic acid. This agent is, however, discountenanced by some practitioners on account of the possible deleterious effect it may have on the pulp ; this objection is not generally sustained. The essential oils are perhaps the most effective agents for use in this connection. The oils of cloves, of gaultheria, cinnamon, thyme, and menthol are all extensively used and are all effective. Of these, thymol acts most promptly ; a saturated solution in alcohol being used, sealed in the cavity with temporary stopping. It is, in addition, a germicide of sufficient activity to sterilize the dentin covering the pulp. The essential oils act as sedatives, and the non-conducting temporary stopping secures rest by preventing the conduction of thermal stimuli. In from twenty-four to forty-eight hours the tooth is placed under the rubber-dam, and excavated (see Chapter XVI.) ; its walls are varnished, and over the wall nearest the pulp a disk of softened gutta-percha is laid. Over this zinc-phosphate paste is flowed. It is usual to complete such fillings with zinc phosphate or gutta-percha, to remain for six months or a year. The conductivity of zinc phosphate is too high to be used as the sole material over pulps which have been the seat of hyperæmia. If the pulp be exposed, it is capped as described in Chapter XVI.

In cases of abrasion or erosion carbolic acid is applied ; an excavation having a retentive form is made, which is varnished and filled with zinc phosphate. A tooth containing a large metallic filling must have the filling removed and after reducing the hyperæmia a non-conducting layer must be placed between the pulp and the filling. The precaution should always be taken, when the pulps of teeth in which cavities have been prepared respond unduly to the temperature-test, to cover the dentinal walls with a layer of non-conducting material. In the absence of this precaution the constant overstimulation of the pulp by thermal

impulses conducted through the metallic filling, may at any time result in hyperæmia.

Idiopathic hyperæmia occasionally affects teeth in which there is no loss of enamel or dentin; and when this condition occurs, it leads to suspicion that the pulp is the seat of nodular deposits. Such teeth are to be dried, heavily varnished, and wedged upon both sides for twenty-four hours, until a gutta-percha cap can be fitted to them completely enclosing the crown. The cap is to remain and to be renewed until the tooth responds normally to the temperature-test.

VENOUS HYPERÆMIA OF THE PULP.

Pathology and Morbid Anatomy.—Considering the mode of vascular supply to the teeth, arteries entering and veins leaving the tooth by a rigid and constricted channel, it is evident that if the arteries be dilated the veins must suffer more or less compression, causing a

FIG. 274.

Section of hyperæmic pulp, showing aneurismal dilatation of the vessels, extravasations of blood, and red blood-disks escaped apparently by diapedesis: *a, a*, dilated vessels; *b, b, b*, extravasated blood. Besides this, red blood-disks are plentifully distributed everywhere in the neighborhood of the veins. The tooth was extracted during a paroxysm of pain. (Black.)

mechanical obstruction to the return of the blood—*i. e.*, venous or mechanical congestion is likely to be established.

In teeth having more than one root the venous engorgement may be

lessened by escape of blood through a second root. In single-rooted teeth congestion *must* be established when the artery or arteries are affected near the apical foramen. It is inferred that in the foregoing conditions described the interference with venous return has been but partial.

The condition raises the tension of the blood in the capillaries and minute veins, and produces stagnation in the emergent veins; there is mechanical stagnation (Fig. 274). It is evident that if this condition continue for a length of time that thrombosis must occur in one or more veins.

Black[1] has described sequelæ of active hyperæmia which appear to correspond with those conditions. Extravasations of red blood-corpuscles occur in the tissues. Œdema, the usual accompaniment of venous congestion, cannot occur, as there is no room for exudations. The condition, corpuscular extravasation, corresponds with that of hemorrhagic infarct—the degeneration and death of more or less pulp-tissue are inevitable. Black suggests that, no doubt, many cases of pulp death *en masse* are due to the condition of general infarction. The force of this suggestion is evident when it is remembered that the arteries to single-rooted teeth are virtually terminal arteries. If the infarction be incomplete, more or less inflammation of the pulp is almost sure to supervene. Disintegration of the red corpuscles may occur and the coloring-matter of the corpuscles may be diffused through the dentin, giving it a pink discoloration. The infiltrated dentin may then become progressively discolored through the characteristic changes of color noted in connection with gradually decomposing hæmoglobin—becoming brown, blue, and finally blue-black.

Symptoms.—The symptoms of this condition, in the absence of definite data, can only be inferential. When the paroxysms of pain are continuous, instead of temporary—that is, when the pain, instead of temporarily subsiding, maintains a constant intensity for hours, and does not respond promptly to therapeusis, and is accompanied by a sense of fulness rather than sharp agony, a condition of serious venous congestion is inferred. The case from which the illustration is taken had been the seat of intense paroxysmal pain for some hours.

Prognosis.—Perfect recovery from this condition is extremely doubtful, so that if the pulp be not intentionally devitalized and removed, it will undergo degenerative changes. The fact that pulps have remained alive for years, after having been the seat of marked congestion, scarcely warrants the attempt to save so seriously crippled an organ in all cases.

Treatment.—Considering the nature of the anatomical changes, it is

[1] *American System of Dentistry*, vol. i.

doubtful whether the pulp can ever fully recover; so that devitalization and extirpation are usually practised in these cases. Before this is attempted or before any attempt is made at permanent treatment, relief is demanded from the pain. After washing out the cavity with warm alkaline solutions, a warm solution of phénol sodique is admirable; the cavity is cleansed of softened dentin, usually exposing the pulp in the operation. This organ, instead of being pink, is seen to have a purplish hue, and immediately protrudes through the opening of exposure. If the surface of the pulp and the cavity-walls be touched with a strong antiseptic, such as a solution of hydronaphthol in alcohol, and a very sharp probe which has been dipped in carbolic acid be used to delicately puncture the pulp, there is an immediate and free flow of blood which is permitted to continue for some minutes, relieving the vascular engorgement. In five minutes the cavity is syringed with a warm antiseptic solution (phénol sodique will answer), and a pellet of cotton containing a saturated solution of menthol in chloroform may be laid over the pulp, and retained in place by another pellet of cotton. This application is usually more effective than even a saturated solution of cocain hydrochlorid in glycerin. The following day an arsenical application may be made. If the pain subside, and the pulp remain quiet for a week under a temporary stopping, some operators have advocated pulp-capping even in this condition.

Inflammation of the Dental Pulp, or Pulpitis.

Definition.—Inflammation, as pointed out in Chapter IV., is a process to be sharply differentiated from that of hyperæmia. It is entirely separate and distinct. While several of the phenomena of arterial hyperæmia are present in inflammation, they constitute but a part of the process. The essential feature of inflammation is the peculiar aggregation and diapedesis of the white blood-corpuscles. Hyperæmia, no matter of what grade or variety, rarely exhibits this feature, and then but slightly, nor in any degree does it present the same types of exudation or of tissue-change observed in inflammation. Inflammation of the dental pulp is a condition in which the phenomena of the inflammatory process (see Chapter IV.) occur in the dental pulp, their course being modified, as in many other tissues of the body, by the peculiar anatomical surroundings.

Causes.—Nowhere more than in the dental pulp does the force of Metchnikoff's dictum, that all inflammations are bacterial in origin, seem to apply. While it must be admitted that here as elsewhere the vast majority of inflammations are bacterial in origin, and that the theory of phagocytosis is the most inclusive that has yet been offered, yet there are numerous conditions in which the bacterial origin has not

been made out. We may accept, however, that inflammation is essentially Nature's method of ridding herself of irritants, and that phagocytosis is the mechanism through which this is accomplished, and leave the question of the necessary association of bacteria *sub judice*.

Taking this position, inflammations of the dental pulp may be divided into infective and non-infective. There are in all probability other irritant factors, in addition to those causing hyperæmia, necessary before inflammation can result. The irritants relate to injuries either through the operation of physical forces or chemical agencies. The chief of the physical forces is the presence of foreign bodies, either upon the surface of the pulp or in its substance. The chemical bodies are those which cause death of the tissues of the pulp, the inflammation being a reaction representing an attempt of the pulp to segregate or expel the dead tissue; or those chemical bodies produced by bacteria which attract the white blood-corpuscles (positive chemotaxis).

Pulpitis is classified, according to its extent, into partial and complete; according to its duration, into acute and chronic; according to its infective character, into purulent and non-purulent; and, again, according to the character of the degeneration which follows upon the inflammatory process. While pathologically these conditions may be clearly differentiated from one another, they may be reduced to more compact groupings according to their clinical significance. For example, acute pulpitis is frequently infective, partial and purulent; chronic pulpitis is frequently non-infective, extensive, non-purulent, and indicative of secondary degenerations.

For the sake of convenience, pulpitis will receive a clinical division into acute and chronic. Some of the chronic varieties have been described under the head of degenerations; others are included in the suppurative diseases of the pulp.

ACUTE PULPITIS.

Causes.—The causes of acute pulpitis are direct and indirect, intrinsic and extrinsic; the vast majority of cases being due to extrinsic causes. The direct intrinsic causes are hemorrhagic extravasations accompanying venous congestion, pulp-nodules, and injury of the vessels at the apex of the root. The direct extrinsic causes are, perhaps, invariably associated with bacterial invasion, a possible exception being the pressure of filling-material upon a thin elastic lamina of softened dentin, covering the pulp. The dental pulp is intolerant of the slightest pressure, and rebels vigorously when subjected to compression. It is not necessary[1] that the pulp should be exposed to permit bacterial infection, and direct or extensive bacterial invasion is probably not

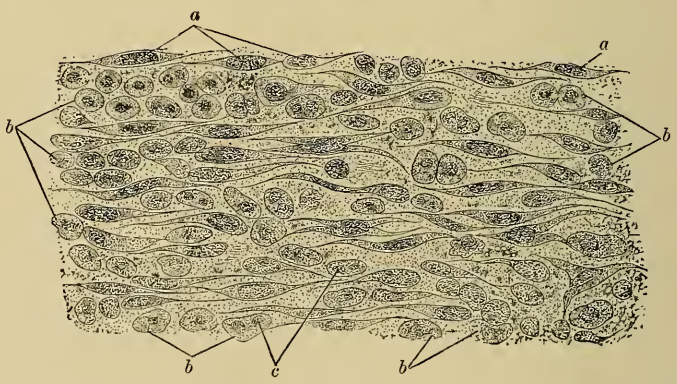
[1] Miller, *Dental Cosmos*, 1894.

necessary for the production of pulpitis. The waste-products, ptomaïns, etc., of bacteria may find their way to the surface of the pulp *via* the dentinal tubuli, through a layer of softened dentin, and excite inflammation. It is extremely probable that infection of the pulp is an invariable consequence of its exposure; but as a pulp may be exposed without subjective evidences of hyperæmia or inflammation, it follows that infection does not necessarily imply inflammation. The presence of a gross irritant, such as a mass of food-débris, vegetable seeds, bread-crumbs, etc., in contact with the pulp will precipitate an acute inflammation in which bacterial relations must be taken into consideration.

"The severity of the inflammation does not appear to be proportionate to the number of bacteria present, and in a highly inflamed pulp we may be able to find but few bacteria. . . . The conclusion seems to be justified that the inflammation is due to the combined action of the bacteria and their products (acids, ptomaïns, etc.) with which the carious dentin becomes impregnated."[1]

The general indirect intrinsic cause of pulp-inflammation may be regarded as active hyperæmia. This condition furnishes a predisposition to active inflammation, as shown in Chapter V. Pulpitis frequently occurs as a sequel to active hyperæmia, and the causes producing this condition must, therefore, be also regarded as the causes of inflamma-

FIG. 275.



Inflammation of dental pulp: *a*, *a*, normal cells; *b*, *b*, *b*, *b*, inflammatory elements; *c*, cells in process of division ($\frac{1}{10}$ in.). (Black.)

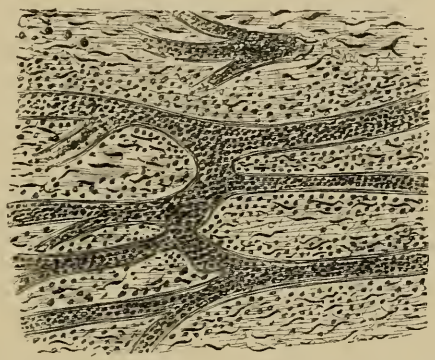
tion. The use of irritating drugs in proximity to or in contact with the pulp may excite inflammation.

Morbid Anatomy and Pathology.—In determining the existence of pulpitis, no matter what the symptoms which have presented or the

[1] Miller, *Dental Cosmos*, 1894.

condition as to exposure, etc., the microscopic examination of sections of the affected organ constitutes the only test; if the changes characteristic of inflammation be absent, no matter what the symptoms, pulpitis did not exist. The essential feature of the process is emigration of the white blood-corpuscles from the small veins into the intercellular matrix of the pulp. At first the inflammatory elements (leucocytes) are scattered through the spaces between the pulp-cells (Fig. 275);

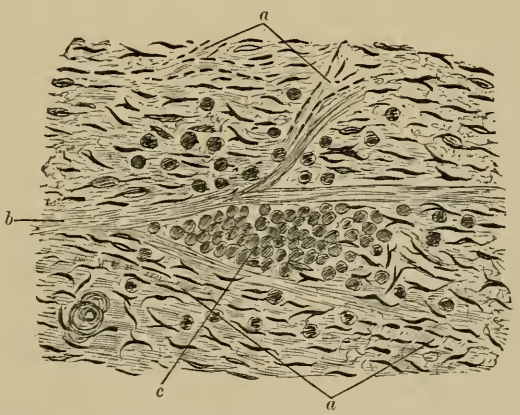
FIG. 276.



Section of dental pulp, showing the invasion of the inflammatory process along the course of the veins—the diapedesis of the white blood-corpuscles. (Black.)

at a later stage the territory is occupied by round indifferent cells alone. The inflammation may be widespread, as shown in Fig. 276, or may

FIG. 277.



Minute inflammatory focus within the tissues of the pulp: *a*, *a*, arterial twigs; *b*, a nerve-bundle; *c*, collection of leucocytes. (Black.)

be localized to some portion of the pulp, as one horn of a pulp; Black noted also inflammatory action occurring in small islands (Fig. 277).

Swelling of the pulp—exudation—cannot occur unless there is a

break in the wall of the pulp-chamber through which additional space can be gained. Black has recorded that "he found *beneath* the layer of odontoblasts in the region of an exposure an unmistakable deposit of inflammatory lymph. The case had a history of severe toothache for two days, two weeks previously. The pulp exhibited evidences of previous extravasations of blood from hyperæmia."

There is evidence that the pulp may recover from attacks of inflammation, and that resolution occurs. In some cases, as shown under the head of calcareous degeneration, the tissues may become infiltrated with calcic material. In others, chronic degenerative changes—inflammatory degeneration—may supervene.

The cases thus far described have been given as non-infective, simply because their infective character has not been clearly made out, although it is very probable that they are infective.

Suppuration of the pulp is a common accompaniment of pulp-inflammation; this being necessarily infective, will be described separately.

Symptoms.—The symptoms of pulpitis are largely a matter of inference. The existence of hyperæmia and a general paretic state of the bloodvessels are judged by symptoms. Throbbing pain referred to the region of some one tooth, and at times referred definitely to the affected tooth, is regarded as a guiding symptom. The tooth may respond to percussion slightly. The pericementum being involved to some extent, the throbbing attendant upon the pulpitis is the reason for the pain being located at times in the offending tooth, instead of being reflected, as usual in pulp-affections. In other cases the pain is referred to far distant points in the course, or at points, of any of the divisions of the fifth nerve. No pain may be felt in the tooth at all, and serious disturbances appear in distant parts, in the eye or ear. The usual symptom, however, in addition to the heavy, throbbing pain in the tooth, is a marked increase of suffering upon assuming the recumbent position. The paretic vessels permit an increased flow of blood into them as soon as its flow to the tooth is favored by gravity. In the upright position during the day the suffering continues, although lessened, as a dull, heavy pain. The pulp responds to both heat and cold, but more to the former than to the latter.

Diagnosis.—Inflammation of the pulp is the usual associate of exposure of the organ whether marked symptoms have been present or not. Its actual existence is judged from presenting the local symptoms noted. A large cavity, with an exposed pulp, with dull, heavy pain, increased in the recumbent position, with little or no response to percussion—pulpitis is diagnosed. If in a tooth containing a large filling these symptoms have been present, pulpitis is inferred.

Pulpitis from injury of the vessels at the apex of the root must be

mentioned. It may occur in consequence of blows, biting upon hard substances, too rapid wedging, the rapid movement of teeth in orthodontia, and the progressive loosening of teeth in pyorrhœa alveolaris. In these cases the pericementum is also affected and the teeth are tender upon percussion. Pain in the teeth upon assuming the recumbent position, dull, heavy uneasiness about the jaws, and inordinate response to thermal stimuli, particularly to heat, point to pulpitis. No doubt many pulps are destroyed by general hemorrhagic infarct in these cases.

Prognosis.—While it is undoubtedly true that the pulps of teeth which have been the seat of inflammation may recover, that resolution may occur, it is the general experience that they usually degenerate and die; if attempts are made at conservation, supplementary pathological changes occur which result in the death and decomposition of the organ. Months or years afterward the tooth increases in opacity, and if the pulp-chamber be opened, the pulp is seen to have undergone decomposition.

Treatment.—Pulpitis when fully established is at times very obdurate so far as local therapeusis is concerned. The same local measures apply as in venous hyperæmia, local bloodletting when feasible, and applications of obtundents and sedatives. Of all local agents, none appears to furnish a greater measure of relief than applications of a paste of cocain hydrochlorid in glycerin, although saturated solutions of menthol and thymol are both effective. Atropia sulfate, gr. j–℈j, is also markedly sedative. These applications should be sealed in the tooth by means of temporary stopping, being careful that no pressure is made upon the pulp or it will respond vigorously.

Before making any medicinal applications to the exposed pulp the cavity should be freely and repeatedly syringed with warm antiseptic solutions, a 50 per cent. solution of meditrina answers well. Coagulant agents are not used, as the coagulum formed interferes with the action of other remedies. If the tooth contain a large filling, it is removed, at least in part, until access to or proximity to the pulp is attained.

The measures stated will afford partial but not complete relief; general antiphlogistic measures are indicated. Of these, perhaps the most effective is local bloodletting.

Nancrede's experiments [1] have shown that one of the most effective methods of unloading an area of engorgement, is to take away blood, and establish a rapid flow through veins adjacent to the area. Making a few cuts with a sharp lancet in the gum overlying the root of the affected tooth, and promoting the flow of blood by holding warm water in the mouth, is a useful measure.

[1] Warren's *Surgical Pathology and Therapeutics.*

Carefully drying the gum and painting over a small area cantharidal collodion and forming a blister is a useful means of derivation.

The continued application of a pepper bag to the gum, and painting the gum with tincture of iodin, both tend to unload the engorged pulp; but none of them has so pronounced effect as direct local bloodletting. If the patient's repugnance to the operation can be overcome, leeching is an effective means of relief. The gum is scrubbed clean, the leech being very loath to attach itself to a dirty surface, and touched with a solution of sugar. The (Swedish) leech, enclosed in a small test-tube, is to be brought to work, by having the tube-mouth pressed upon the gum. As soon as the leech is engorged, the tube is withdrawn slightly and salt (sodium chlorid) is dropped upon the animal, which then falls back into the test-tube.

The administration at night of ammonol, grs. x, in addition to local sedatives and local bloodletting, will usually secure quiet. If the patient be at all costive, a dose of Epsom salt, magnesium sulfate, a tablespoonful in a goblet of water, is an additional means of relief. Quiet of the pulp must be secured before an arsenical application is made, or the latter merely increases the irritation instead of promptly devitalizing.

Suppuration of the Pulp.

Definition.—By suppuration of the dental pulp is meant a formation of pus on its surface (ulceration) or in its substance (abscesses). It occurs both as an acute and as a chronic affection.

Fig. 278.

Invasion of pulp by micrococci. (Arkövy.)

Causes.—The immediate cause of suppuration of the pulp is the ingress of pyogenic organisms to the pulp. As in inflammation of the pulp, while usually associated with direct exposure of the pulp, suppuration may occur in pulps covered by softened, or even unsoftened dentin.[1] "Bacteria which have entered the body through wounds may be deposited in the pulp as well as in any other part of the body, wherever there may be a *locus minoris resistentiæ* at the time.

Arkovy[2] (Fig. 278) first observed infection of the pulp while still covered by a layer of unsoftened dentin. Miller[3] questions whether bacteria can pass through any but very thin layers of dentin. He states[4] that sections of the overlying dentin in a case of suppuration of

[1] Miller, *Dental Cosmos*, 1894.
[2] *Diagnostik der Zahnkrankheiten.*
[3] *Micro-organisms of the Human Mouth.*
[4] *Dental Cosmos*, 1894.

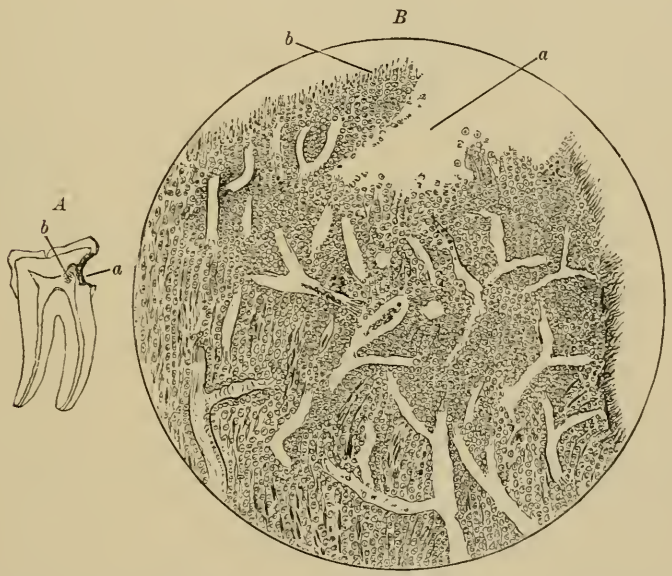
the pulp showed the same forms of bacteria as were found in the pulp itself.

In cases where suppuration has occurred in teeth containing large fillings which are perfect and intact, and the pulp has never been exposed, it is a reasonable inference that the organisms necessary to pus-formation have found their way to the dental pulp *via* the general circulation. This infection in purulent pulpitis is a mixed one, both cocci and bacilli being present, and in later stages of degeneration other forms appear also.

Suppuration of the pulp is a not infrequent sequel of the capping of pulps which had given evidence of a previous hyperæmia or inflammation.

Morbid Anatomy and Pathology.—Anatomically pulp suppuration, purulent or pyogenic pulpitis, is of two general varieties; one begins upon or close to the surface of an exposed pulp, and gradually destroys the organ through a process of progressive (Fig. 279) ulcera-

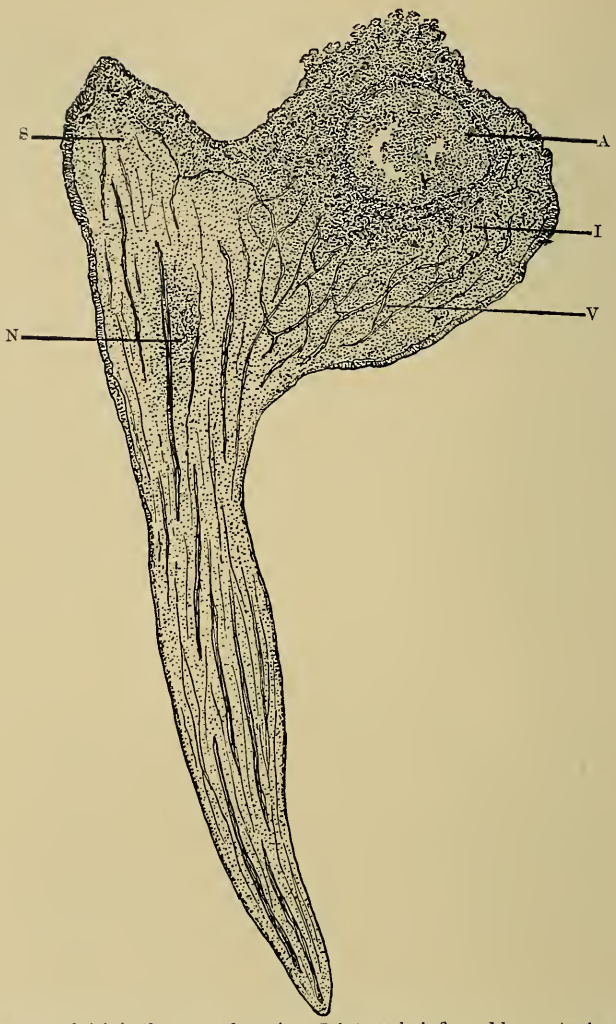
FIG. 279.



A, diagram of lower molar, with caries at *a* which exposes the pulp: the darkened portion at *b* shows the extent of the inflammation; the rest of the organ was free from inflammatory change. *B*, illustration of the inflamed tissue, showing a part destroyed by suppuration at *a*; the odontoblasts are undermined at *b*; the bloodvessels which were filled with blood-clot in the section are left blank here, that they may be more apparent. (Black.)

tion; the second, that confined in the substance of the pulp, causes the gradual destruction of a part or all of the pulp through the formation of circumscribed abscesses (Fig. 280).

Fig. 280.



Acute suppurative pulpitis in the coronal portion: I, intensely inflamed horn; A, abscess; V, blood-vessels engorged with blood; S, superficially inflamed horn; N, nest of inflammation. × 10. (Bödecker.)

Ulceration of the Pulp.

Of these two forms, ulceration is the more common. The capillaries (Fig. 279) are blocked with coagulated blood (they are left open in the illustration to clearly mark their position); the intercapillary meshwork is occupied by inflammatory exudation; the surface of the pulp is eroded, and covered with pus-corpuscles; the ulcerative process is undermining the layer of odontoblasts. The suppurative process penetrates the body of the pulp, following the direction of its veins and hollowing out the organ into a deep cavern. Black regards

the persistence of the layer of odontoblasts as indicating an inferior vitality, as it shows they are less susceptible of change of form than the other cells of the organ.

The process of ulceration may continue for weeks or months until the entire organ has been destroyed molecularly. The necrotic portions undergo putrefactive decomposition, probably passing through the same stages that any albuminous substance passes in its serial decomposition, into the end-products—ammonia, carbon dioxid, hydrogen sulfid, and water.

"Very interesting and instructive results were obtained by examining material from different parts of the same tooth. In the case illustrated in Fig. 281 the pulp-chamber at *a* was wide open and filled with food-particles, which had a foul, half-putrid odor; at *b* the pulp was putrid and foul-smelling; at *c* there was a small abscess, filled with pure white pus, while the tissue between this point and the apex of the root was highly inflamed and bright red. Material from the pulp-chamber (*a*, Fig. 281) contained the forms shown in Fig. 282; material from point *b* those shown in Fig. 283, and from the point *c* those shown in Fig. 284. We perceive a gradual diminution of the large cocci, and the appearance of small, delicate cocci and diplococci" (Miller).[1]

FIG. 281.

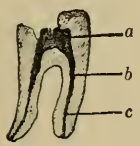


FIG. 282.

FIG. 283.

FIG. 284.

FIG. 285.

Micro-organisms found in cultures from a gangrenous pulp.

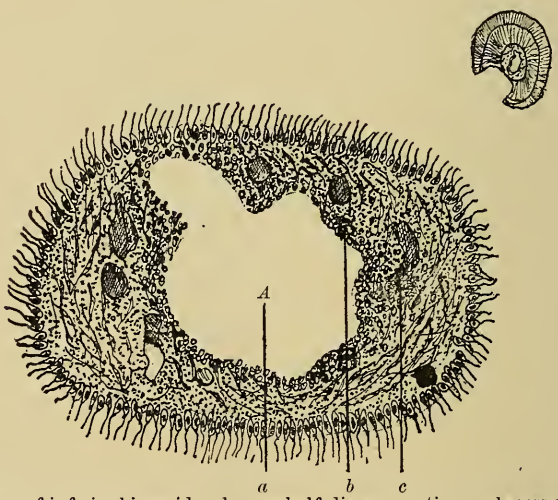
ABSCESS OF THE PULP.

Abscess of the pulp is usually situated near the point of exposure of the organ. It may be confined to one horn of the pulp, or may

[1] *Dental Cosmos*, 1894.

involve nearly the entire substance of the pulp, the peripheral tissue of the pulp being unbroken. Abscess may exist at some distance beneath the surface of the pulp, and the latter be still covered with a layer of dentin. The writer once uncovered the horn of a molar pulp which was covered by a lamina of hard dentin, and no fluid appeared; but upon passing a sharp probe into the *white* area of exposure for over one-eighth of an inch or more there was a free flow of pus which quickly filled the larger carious cavity. A pulp removed entire from a tooth which was yellowish-white in color and unbroken showed upon section its interior hollowed out into an enormous abscess-cavity (Fig. 286). The bloodvessels were blocked; the peripheral tissues were unal-

FIG. 286.



Transverse section of inferior bicuspid pulp, one-half diagrammatic: *a*, abscess-cavity; *b*, embryonic cells at the periphery of abscess-cavity; *c*, occluded bloodvessels.

tered; between the odontoblasts and the abscess-cavity, the latter lined with pus-corpuscles, evidences of inflammation were plenty. Black found that the odontoblasts retained their form after neighboring cells of the pulp had been destroyed.

Miller's[1] researches show a preponderance of cocci and micrococci in cases of enclosed abscess; cocci and diplococci were of constant occurrence. Many of the forms, both cocci and bacilli, were cultivable upon gelatin and agar-agar. Some of them, cocci and bacilli, brought about the liquefaction of gelatin; others did not. So that it must be inferred that infective inflammation and necrosis of the pulp may occur without suppuration. In some instances streptococci were found. In the freely exposed pulps varieties of organisms were found which would render clear the possibility of a general infection by way of the dental pulp.

[1] *Dental Cosmos*, 1894.

Symptoms.—Many cases of ulcerative suppuration of the pulp may run their course to complete or almost complete destruction of the organ, and no uneasiness arise until septic pericementitis appears, which it almost invariably does some time subsequent to death of the pulp. In other cases, however, pains characteristic of inflammation of the pulp are noted, but response to the cold test has almost disappeared. Intense pain may exist when the pus does not find ready exit owing to food-débris being massed in the cavity of decay or to the presence of a large filling. The condition then resembles that of abscess of the pulp. The usual history of the latter disease is as follows; in a tooth containing an enormous filling, one in which the pulp has been exposed, or in a tooth having a large carious cavity, the patient gives a history of discomfort or decided pain, appearing at intervals; the existing condition having been ushered in by dull, gnawing pain, which is usually not positively located, although it may be. The pain grows in intensity, and, in contradistinction to the pulp-conditions previously described, pain is relieved instead of increased by applications of cold. It may be, however, that the prolonged contact of ice-water may induce a response. The response to heat is marked, so that a mouthful of hot coffee may precipitate an attack of severe and continued pain. If the pulp be freely exposed and pricked with a sharp instrument, a flow of pus follows in many cases, and the relief is almost immediate. In the earlier stages a period of throbbing pain may follow evacuation of the pus.

In other cases the response to heat may decrease until it is almost absent, and the case only be seen when evidences of the action of bacterial products upon the pericementum appear, which they usually do in the later stages of pulp suppuration; when the tooth becomes loose, extruded, and tender upon percussion. If untreated, symptoms of pulp and pericemental disturbance may disappear for weeks or months; but if the parts be not perfectly sterilized and reinfection prevented, it is only a question of time when septic pericementitis will arise.

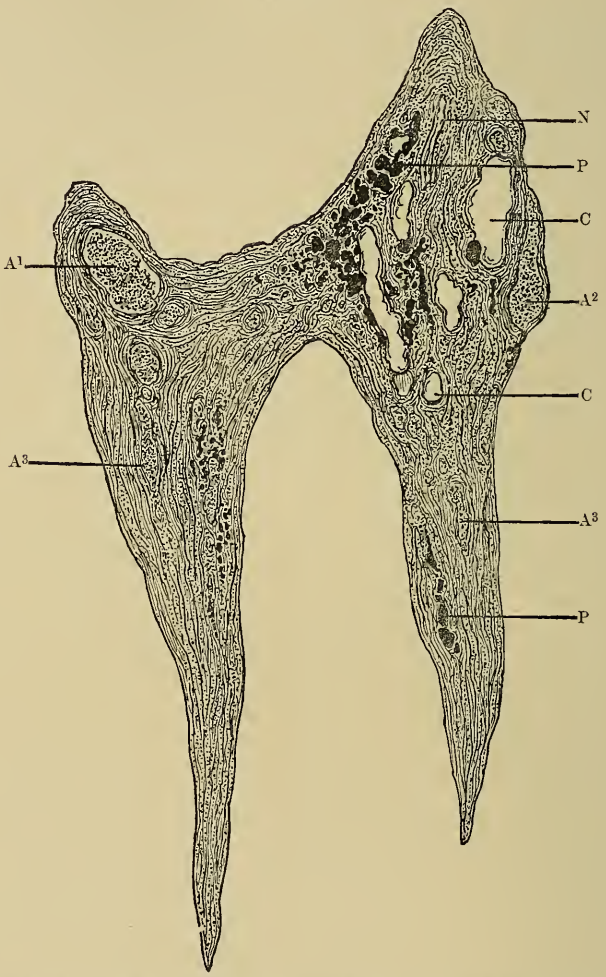
Diagnosis.—The most valuable diagnostic sign is the peculiar reaction to thermal stimuli—the decreasing, then absent response to cold, and the increasing reaction to applications of heat. This reaction, together with the continued gnawing, and full sensation in the tooth, usually affords, a diagnosis, which is confirmed by evacuating pus from the pulp.

Prognosis.—General experience regards ulceration and abscess of the pulp as precursors of the death of the organ. Usually this is by progressive suppuration. It is undoubtedly true, however, that attempts at circumvallation of the dead tissue are made in some cases (Fig. 287). The pus-cells undergo degeneration and the abscess-site may be the seat of calcareous deposits. Even in these cases death is

delayed, not averted. The remainder of the pulp undergoes atrophic changes, and commonly suppuration reappears.

Treatment.—The treatment of the case consists in relieving the existing pain, completing the devitalization of the pulp, and removing

FIG. 287.



Chronic suppurative pulpitis terminating in calcification of the pus and atrophy of the pulps. A^1, larger abscess, filled with calcified pus; A^2, abscess at the periphery of the pulp; A^3, A^3, small longitudinal abscesses, all calcified; N, calcified nerve-bundle; C, C, calcareous depositions in the fibrous pulp-tissue; P, P, pigment-clusters from previous hemorrhage. × 10. (Bödecker.)

it in such a manner that no organisms or dead matter are carried beyond the apex of the root.

To secure relief, evacuation of the pus is imperatively necessary. The organ is freely exposed, exercising no pressure in gaining free access to it. If pus do not flow upon exposure of the surface of the pulp, the

cavity and pulp, the tooth being under rubber-dam, are drenched with strong antiseptics—a spray of hydrogen dioxid or of meditrina—and a sharp, slender sterilized probe is quickly passed into the substance of the pulp, when if pus be present it will usually escape freely through the opening thus made.

If the pus-formation be limited and circumscribed, throbbing pain may follow, which promptly quiets under an application of cocain in glycerin. The application is not made until the pus-flow ceases. A pellet of cotton wet with a 3 per cent. solution of formalin, or a saturated solution of thymol, is laid upon the pulp and the cavity is sealed for twenty-four hours (never longer), and then an arsenical application is made. Should the exposed portion of the pulp be insensitive it is burred away until access is had to the vital portion, where the arsenic is to be applied. Antiseptics are to be freely used, and the rubber-dam applied before entrance to the pulp for any therapeutic applications.

CHAPTER XIX.

CHRONIC DEGENERATIONS AND DEVITALIZATION OF THE PULP.

Some of the chronic degenerations of the pulp have been described under the head of secondary deposits in the pulp-substance. The grades of hyperæmia, active inflammation and suppuration represent the acute degenerations of the organ.

Chronic Inflammation.

As stated in Chapter XVIII., ulceration of the pulp commonly pursues a chronic and progressive course until the organ is destroyed; the condition may, therefore, be termed chronic purulent pulpitis (ulcerosa), as an inflammatory zone lies beyond the line of suppuration. This condition has already been discussed.

Abscess of the pulp may pursue a chronic course, as stated. Attempts are occasionally seen at repair in the affected pulp, the abscess-area being marked off, and the elements contained in it becoming the seat of calcareous deposits.

SCLEROSIS OF THE PULP.

Inflammation of a low grade may persist in the pulp for long periods, giving rise to an increase of its fibrous tissue with atrophy of the pulp-elements, producing a condition similar to that found in chronic interstitial inflammation in some other tissues—a sclerosis. Instead of the usual distribution of myxomatous tissue, bands and bundles of fibrous tissue appear. The pulp appears shrunken and stiff, bloodvessels are contracted, and the nerve-fibres have undergone partial or complete atrophy.

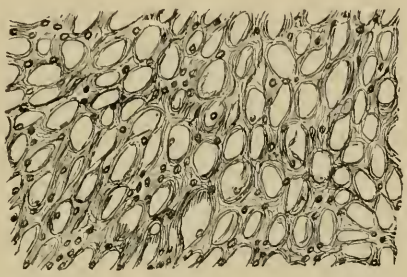
Black[1] found that in the late stages of sclerotic atrophy areolæ developed in the bundles of connective tissue, the inflammatory elements having disappeared, and the areolæ being occupied by fluid. Arkovy describes this condition as reticular atrophy of the pulp (Fig. 288). The condition would point, as suggested by Black, rather to venous hyperæmia as the cause of the œdema than to inflammation; but the evidences of former chronic inflammation in the existence of the bundles of reticulated tissue show this to have been the essential condition. The

[1] *American System of Dentistry*, vol. i.

observations of the same writer indicate that atrophy of the odontoblasts is a usual accompaniment of all of the chronic pulp affections.

Sclerotic and other chronic degenerations of the pulp usually present the history of one or more attacks of pulpitis in the past, with

FIG. 288.

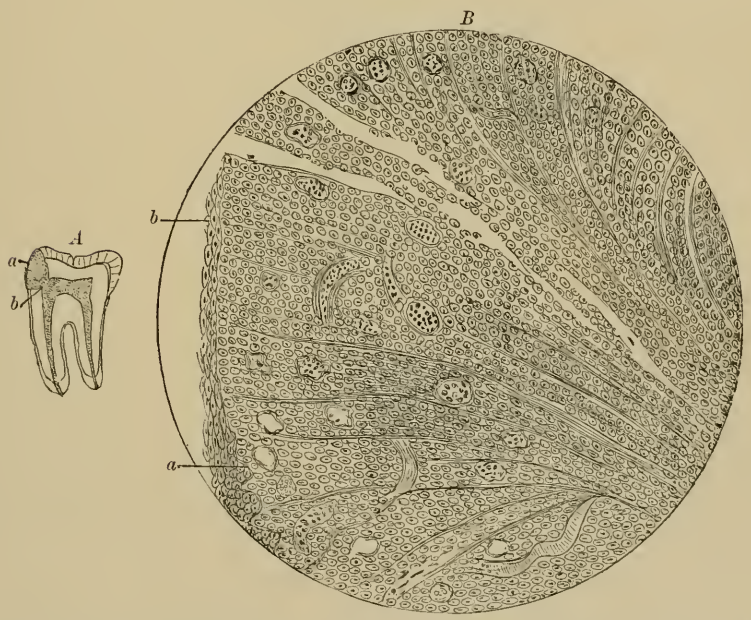


Chronic inflammation of the pulp, areolation, and degeneration. (Black.)

more or less continuous uneasiness extending over a long period. The response of the pulp to all tests becomes diminished and dull.

Treatment.—Such pulps are to be devitalized and removed.

FIG. 289.

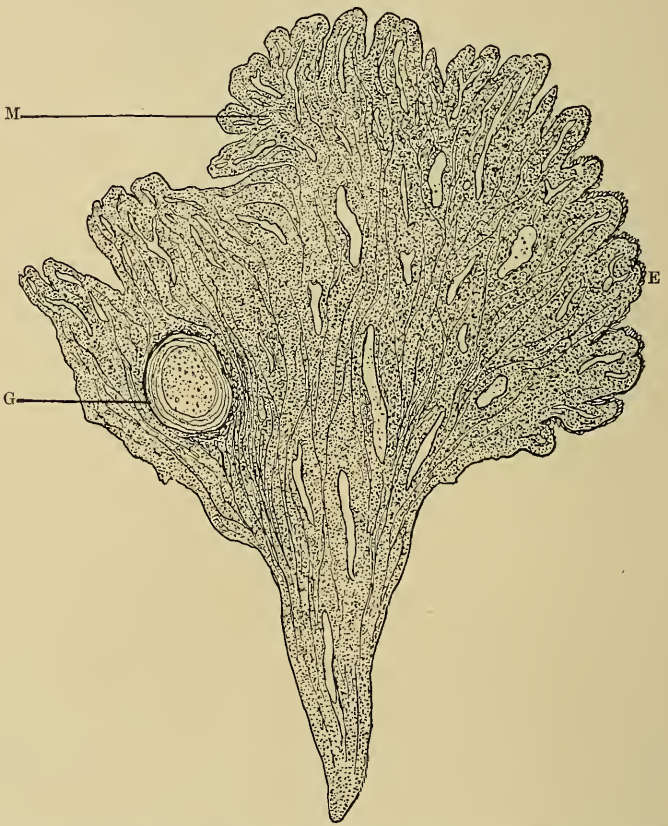


A, a first lower molar with a cavity at *a* completely filled by a hypertrophy of the pulp, which has grown out through the orifice, exposing the pulp at *b*. *B*, a field illustrating the tissue of the growth, which is composed almost entirely of granulation-tissue of a very primitive type: *a*, a covering of epithelium presenting papillæ; *b*, epithelium apparently without papillæ. (Black.)

CHRONIC HYPERTROPHIC PULPITIS.

When the pulp is exposed over a wide area, long-continued chronic inflammation may lead to an enlargement of the organ with a protrusion of altered pulp-mass through the orifice of exposure, producing the condition known clinically as fungous pulp. When the growth

FIG. 290.



Hyperplastic myxomatous pulp, which filled a carious cavity: M, lobules made up of papillæ of a myxomatous structure, rich in capillary and venous bloodvessels; G, calcareous globule: E, epithelial cover of papillæ. × 10. (Bödecker.)

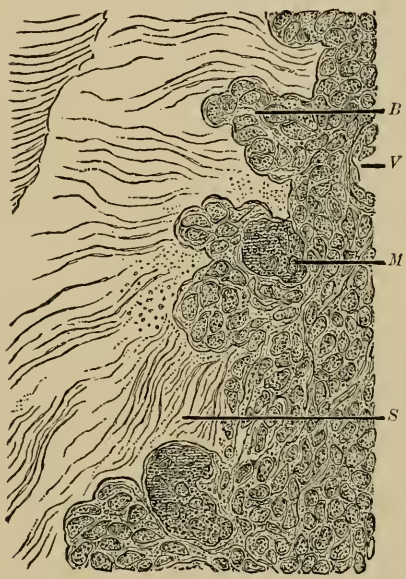
extends beyond the boundaries of the orifice and then increases in bulk it forms a pedunculated mass to which the term polypus of the pulp has been applied.

Morbid Anatomy and Pathology.—The growth has its origin in a chronic inflammation of the body of the pulp; the organ swells, and contact with the sharp edges of the orifice of exposure excites a continued irritation leading to further proliferation of the cells of the inflamed part, so that a large mass of embryonic tissue is formed

(Fig. 289), termed by Black granulation-tissue of a low type. As in the granulation-tissue of repair, bloodvessels grow into this mass (Fig. 290), so that it may bleed at a slight touch. Black noted in his case illustrated, a covering of squamous epithelium upon the periphery of the growth, which might be interpreted as the transformation of mesoblastic into epiblastic tissue, but the correct explanation beyond doubt is that advanced by the same author, that the epithelium is transplanted from the gums, and grows after the manner of a skin-graft.

These growths may undergo further changes; higher organization of the granulation-tissue occurs and fibrous tissue is formed; the cells may undergo degenerations, first granular, then fatty, and suppuration and

FIG. 291.



Acute pulpitis: *S*, secondary dentin; *B*, bay-like excavations filled with medullary or inflammatory corpuscles; *V*, transverse section of a bloodvessel; *M*, multinuclear body. ×300. (Bödecker.)

gangrene may occur. Tomes[1] records a case where calcification of a hypertrophied section of a pulp occurred; but as the case was due to traumatism (fracture of a tooth), different vital conditions existed from those in the cases under discussion. Actual calcification of the mass is scarcely possible, although calcareous degeneration may occur (see Chapter XVIII.).

Resorption of the walls of the pulp-chamber may occur as an accompaniment of chronic pulpitis. What appears to be an idiopathic dentin-resorption is described in Chapter XVII. Black records a case

[1] *Dental Surgery*, 3d ed.

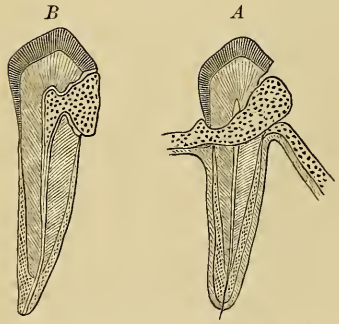
where after pulp-capping in a lower molar and the insertion of a large gold filling the tooth was examined at the end of ten years; for two or three years the pulp had given evidences of irritability, and when the pulp was removed the pulp-chamber was found enormously enlarged and opening into the pericementum between the roots of the teeth. Fig. 291 exhibits resorption of previously formed secondary dentin with the probable agency through which the resorption is brought about. The area of resorption is invaded by numerous multinucleated cells, which are evidently performing the function of odontoclasts.

Symptoms.—The symptoms of chronic pulp inflammations and degenerations are usually those of long-continued discomfort, with reflex pains, which rarely persist into the latest stages of degeneration. The response to heat and cold, present at first, declines until the pulp scarcely reacts, and then but slowly.

No nerve-fibres develop in the hypertrophic pulp-tissue, so that the new growth has no sensitivity in itself, although pressure upon it may cause sharp pain through the still vital pulp-nerves themselves.

Diagnosis.—The only condition with which hypertrophic pulp may be confounded is a pedunculated growth of gum-tissue through a cavity at the neck of a tooth beneath the gum-margin. It is important to differentiate between these conditions, because if an application of arsenical paste be made to a fungous gum, the destruction of tissue may extend into the sound pericementum. The physical appearances of the two are alike: they both bleed freely and have about the same degree of sensitivity. If the degenerative changes have not involved the entire substance of the pulp, a reaction to cold may be secured, which will, of course, determine the diagnosis. After isolating the tooth under rubber-dam a spray of ethyl chlorid is directed against the polypus, and when it is entirely insensitive, a sharp blade is passed entirely around the periphery of the carious cavity detaching the fungous mass. The source of the tumor may then usually be clearly seen. If any doubt exist, the cavity is freely syringed with antiseptics, such as pyrozone or meditrinia; a pellet of cotton saturated with the same is inserted and over it temporary stopping is firmly packed. If the pulp be the seat of the growth, it may rebel against the pressure, and require a less firm dressing, so that slight reaction to the pressure is to be regarded as evidence that the growth arises from the gum.

FIG. 292.



A, hypertrophy of gum similating fungous pulp, *B*.

Treatment.—Devitalization and extirpation of the pulp.

DEVITALIZATION AND REMOVAL OF THE DENTAL PULP.

It will have been noted in the immediately foregoing chapters, that the intentional devitalization and removal of the dental pulp are demanded as an indicated therapeutic measure in pulp diseases which tend to self-destruction of that organ. The one universal method of accomplishing the death of the pulp *en masse* is by applications of arsenic trioxid—arsenious acid. Although other means have been suggested and adopted, none is so certain and effective as this one. After freely exposing the organ to be destroyed, a general anæsthetic (nitrous oxid) has been administered, and the pulp removed while the patient is in the anæsthetic state. Sprays of rapidly vaporizable substances, such as ethyl or methyl chlorid, directed against the exposed pulp, the tooth being isolated under rubber-dam, will in many cases render the pulp entirely insensitive, although, as a rule, they fail to entirely anæsthetize to the apical foramen. Applications of even saturated solutions of cocain being ineffective, it has been suggested to inject cocain into the pulp: the surface of the pulp is benumbed by applications of strong solutions of cocain, the needle of a hypodermic syringe containing a solution of cocain hydrochlorid (from 4 per cent. to 10 per cent.) is quickly thrust into the pulp-canal, and a drop of the solution forcibly injected; in a few seconds the pulp may be so benumbed that it may be removed.[1] This procedure, however, appears to fail as often as it succeeds.

Cocain cataphoresis is usually effective, although in conditions of active hyperæmia and inflammation even the maximum current and saturated solutions of the alkaloid may fail to subdue the irritability of the pulp. The pulp may be destroyed piecemeal by applications of strong caustics, such as zinc chlorid or chromic acid and sodium or potassium hydrate, but their action is slow and the operation tedious; moreover, it is not without danger; in addition, the application of these agents in sufficient strength is usually followed by severe paroxysms of pain.

Arsenious acid—arsenic trioxid—is prompt, certain, and complete in its action and has maintained its position in dental therapeutics since introduced by Spooner for this purpose in 1836. The progenitor of all present arsenical pastes was the formula of J. D. White, given some forty years ago:

℞. Arsenious acid, Morphiæ sulph., āā
Carbolic acid, q. s. ft. paste.—M.

[1] Maxfield, Proc. *New Jersey State Dental Society*, 1894.

This was followed twenty-five years ago by that of J. Foster Flagg:

℞. Arsenious acid,	gr. x;
Morphiæ acetat.,	gr. xx;
Ol. carophyllum,	q. s. ft. paste.—M.

The advantages of the latter were demonstrated clinically, and within a few years scientifically. The purpose and effects of the ingredients of arsenical pastes will become more evident after an examination of the effects of arsenic upon the pulp.

Effects of Arsenic on the Pulp.—Since the introduction of arsenical preparations definite physiological effects have been noted following their use. First, a grumbling pain, rising in a few hours to acute paroxysmal pain; then a gradual and in some cases a sudden cessation of pain, after which the pulp fails to respond to applications of cold.

These effects were explained[1] as follows: "A *minute* portion of the arsenic being introduced into the pulp-circulation, acts as a dynamic, vital irritant, causing determination of blood to the part" (*i. e.*, arterial hyperæmia), and gives rise to the throbbing pain. Congestion (presumably venous hyperæmia) follows, occasioning the cessation of throbbing and the appearance of dull, gnawing pain in the tooth. "The death of the pulp *en masse* is due to strangulation of the vessels at the apex of the root in consequence of the congestion."

The subsequent decomposition of the pulp—putrefaction—was deemed due to the amount of arsenic absorbed by the pulp prior to its death being insufficient to preserve it. Flagg states[2] that tests of pulp which have been devitalized by arsenic show but an infinitesimal amount of the agent to be present in them.

The same writer points out the existence of pericemental irritation in the last stages of pulp-devitalization; that is, in from four days to a week after the application; regarding it as not due to the irritation produced by the arsenic, but to an extension of the vascular condition from the pulp; the pericemental disturbance is limited, ceasing in a few hours or a day or two.

Arkovy[3] was the first to point out the details of the action of arsenic upon the dental tissues:

"1. As_2O_3 brought into contact with the tooth-pulp acts in the following way: a certain degree of inflammatory hyperæmia, total or partial, depending upon the quantity of the agent applied, sets in; the bloodvessels become expanded, and here have a tendency to thrombosis. This latter effect may also be in connection with embolism of the capillaries, when the agent is quickly taken up into the bloodvessels.

[1] J. Foster Flagg, *Dental Cosmos*, 1877. [2] *Ibid.*

[3] *Transactions Internat. Med. Cong.*, London, 1881.

"2. As_2O_3 produces no coagulation of tissue whatever.

"3. It has a specific influence upon the blood-corpuscles, combining with the hæmoglobin to form a compound of arsen-hæmoglobin, and of this chemical process there seems to be evidence in the profuse yellowish tinge of the whole pulp-tissue and in the discoloration of blood in several of the bloodvessels.

"4. In nearly every case it is taken up *in substantia* (in form of molecules) into the blood-ways; when there it produces, besides the above-mentioned changes, granular detritus of the contents and anæmic collapse—shrinkage, the latter effect being brought about nearly exclusively in cases where greater doses were used.

"5. The bulk of the pulp-tissue—viz., connective-tissue fibres and odontoblasts—undergoes no change whatever; not so the connective-tissue cells, which increase three or four times their normal size.

"6. The special action of arsenic trioxid upon the nerve-elements consists in the following: the neurilemma is only so far influenced that its nuclei are somewhat increased; a more essential change takes place in the axial part, where, after the application of more than one mgrm. granular destruction of myelin sets in, and the axis-cylinder commences here and there to disappear. A very surprising alteration may be seen in the notchy tumefaction of the axis-cylinder, described heretofore almost only in cases of central lesions.

"7. All these alterations occur in and among normal-looking tissue.

"8. The action of arsenic trioxid is macroscopically exhibited by a brownish-red tingeing of the whole or of certain parts of the pulp-body, as well as of the neighboring dentin and the cementum, this latter in cases treated with greater doses—viz., two to five mgrms. This alteration is most expressed at the top of the crown-pulp and at the apical one-fourth to one-third part. This circumstance may be considered as an external evidence of the devitalization being completely attained to."

In some cases the pinkish discoloration of the dentin may be marked; the broken-down corpuscles of the extravasated blood have their coloring-matter taken up by the odontoblasts, and being distributed through their protoplasmic processes produce a condition technically known as suffusion. The same result may be an attendant upon injury to the vessels from other causes, from sudden thrombosis, as when teeth are moved too rapidly in regulating.

Miller's experiments[1] upon the tails of mice, made without and with rings at the root of the tail to simulate the surroundings of the apical vessels of a tooth; made without and with encasement of the tails in plaster-of-Paris to imitate the rigid surroundings of the dental pulp:

[1] *Dental Cosmos*, 1894.

showed that in the absence of the plaster encasement, enormous œdema of the tail was produced and a sensory paralysis of the hind limbs; complete anæsthesia of the tail occurred in forty-eight hours. "The action of arsenic appeared somewhat accelerated when a glass ring was applied close to the root of the tail. In more than forty cases there was not one in which the action of the arsenic extended beyond the ring, and the action was not appreciably affected by enclosing the tails in plaster casts. The action of the arsenic is of a progressive nature, beginning at the point of application and extending gradually in each direction." The indication is, therefore, that a minute portion of the arsenic is taken into the pulp; this may be either through the odontoblastic processes in cases of non-exposed pulps, or, when exposed, by the cells themselves, and causes violent reaction of the vessel-walls, which dilate to their utmost; extravasation of corpuscles occurs and circulation is checked at the end of the root, the pulp-vessels are mechanically occluded, so that no absorbed arsenic is carried beyond the pulp extremity. The arsenic exercises its paralyzant and degenerative influence upon the nerves of the pulp, after first inducing a violent irritation, which may be due to the specific action of the arsenic upon the nerves or to the intense hyperæmia. The strangulation theory cannot, however, hold in all cases, for progressive death of structures free to expand occurs after applications of arsenic, so that the specific and gradual action of the poison must be the important factor. Strangulation would imply an always sudden death of the pulp *en masse*, which evidently does not always occur, for complete death of the organ may be gradual and require several days. In other cases repeated applications may be necessary.

These experiments illustrate the danger of making arsenical applications to immature teeth or those in which resorption of the roots is in progress. If the full constriction of the apical foramen has not occurred, it is possible that a portion of the arsenic may be carried beyond the apex of the root into the pericementum. This danger, it may be remarked, has been recognized for the past thirty years.

Variations in the Action of Arsenic.—In most cases of fully formed teeth in young adults, an application of arsenical paste directly to the exposed pulp will be followed by the complete death of the organ in forty-eight hours. At the expiration of that time a sterilized broach may be passed almost to the apex of the root and the pulp removed *en masse* without pain. If pulp-nodules exist, the action of the arsenic may be delayed or in some cases be almost *nil*. In calcareous and other chronic pulp-degenerations the action is also delayed. If arsenical applications are made over a layer of dentin, the same delay is noted, and is increased in very mature teeth.

Some pulp, irrespective of the pulp condition, exhibits a peculiar idiosyncrasy in resisting the action of arsenic, requiring large doses and a week or longer application before succumbing.

Form in which Used.—Miller[1] has pointed out the influence exerted by the several agents used in conjunction with the arsenic. The constituents of the common prescription,

℞. Acidi arsenosi, }
Morphiæ acetatis, } āā.
Acid. carbolici, q. s. ft. paste.—M.,

were supposed to have the following properties: the arsenic trioxid being the devitalizing agent; the morphia is used to lessen or deaden the pain of the application; the carbolic acid is an analgesic menstruum. Carbolic acid and other coagulants produce an eschar, a coagulum, which delays the absorption of the arsenic, so it should be discarded. It has always been questioned just what extent of analgesic effect morphia possessed; some say none, holding that the lessened pain is due to the reduction of the amount of arsenic applied. Certainly, the painful effects of the poison do appear to be modified by the size of the dose employed. Iodoform, also used as an analgesic ingredient in some prescriptions, is of doubtful value. Cocain, naturally, was made one of the ingredients of arsenical pastes soon after its introduction into the materia medica. The usual prescription at present is:

℞. Acidi arsenosi, gr. x;
Cocain. hydrochlorid., gr. xx;
Ol. cinnamomi, q. s. ft. paste.—M.

Miller suggests using thymol in connection with arsenic, it being both analgesic and antiseptic. He offers the following general rules as deductions from his observations:

1. The rapidity and intensity of the action of arsenious acid depend, under certain circumstances, to a very considerable degree upon the substance or substances with which it is incorporated.

2. Where there is but a small point of exposure, and in particular where extensive calcification has taken place in the pulp, escharotics should be avoided, since the coagulation of the tissue retards the absorption of the arsenic. This retardation is but slight where there is a broad surface of exposure. In stubborn cases, where applications of the ordinary paste fail to effect the devitalization, a paste consisting of arsenious acid in oil of cloves, glycerin, or salt solution should be employed, undiluted by any third constituent.

[1] *Dental Cosmos*, 1894.

3. Thymol is worthy of a trial as a substitute for morphia, on account of its anæsthetic and antiseptic properties.

4. For devitalizing pulps of temporary teeth or remains of pulp-tissue in root-canals, arsenious acid, if employed at all, should be diluted with two or three parts of some other constituent (thymol, zinc oxid, morphia, iodoform).

Cobalt was introduced by Robert Arthur as a devitalizing agent some forty years ago. Within recent years it has been employed, notably by the Herbst method (which see), to destroy pulps. The cobalt paste of Herbst was analyzed by E. C. Kirk[1] and found to consist of *metallic* arsenic and cocain hydrochlorid. Kirk suggests that free acids which cocain salts may contain, or the chlorin from the chlorid, may combine with the metallic arsenic and form soluble salts. Commercial cobalt will certainly devitalize the dental pulp, but it is in consequence of the arsenic contained in it.

Mode of Application.—It is always to be borne in mind that arsenic acts upon all living tissue in the destructive manner that it does upon the dental pulp, so that the first consideration in making an arsenical application is the precise placing and sealing of the paste so that none of it shall come in contact with any vital tissue other than the pulp. Second, in conditions of venous hyperæmia or inflammation arsenic, instead of exercising its actively destructive property, serves but to exaggerate the existing vascular conditions with the attendant symptom, pain. Third, but a minute quantity of arsenic is required to kill a pulp; and if an excess be used, the attendant pain is much increased. Fourth, if any pressure be exerted upon the pulp by the material used to seal in the paste, the pain will be increased in the degree of the pressure. Fifth, in the vast majority of cases the pulp-cavity is the seat of infection, so that careful sterilization should precede the application of arsenic. Sixth, to insure accuracy of placement, freedom from leakage, and sterilization, the application of the rubber-dam should precede the placing of the arsenical paste.

In the vast majority of cases arsenical applications are made directly to the point of exposure in the cavity of decay. In case the gum overhang the cavity-margins, it must be removed until the margins are clear and visible. The cavity is syringed repeatedly with warm pyrozone; and a pledget of cotton saturated with meditrina, thymol (saturated solution), or hydronaphthol (alcoholic solution), and over this a pellet of cotton which has been dipped in sandarac varnish is placed. The dressing must be renewed in twenty-four hours. Coagulating antiseptics should not be used, as they form a coagulum upon the surface of the pulp and interfere with the action of the arsenic. If the cavity be upon the buccal

[1] *Dental Cosmos*, 1893, p. 247.

face of a molar, or in a situation where the gum-tissue has hypertrophied and covers the cervical portion of the cavity as a bulbous mass, the redundant portion may be trimmed away by means of a sharp, curved bistoury or gum-lancet.

As soon as the cervical border of the cavity is clear of the overhanging gum it is syringed with warm antiseptics and the rubber dam adjusted. The softened dentin is freely cut away until the cavity has a retentive form and the area of exposure is clearly outlined. If the pulp be the seat of immediate or very recent hyperæmia or inflammation, anodyne antiseptics should be sealed in the cavity for a day or two before making the arsenical application. An admirable prescription in this connection is thymol or menthol with cocain made into a paste with glycerin; a pledget of cotton dipped in this mixture and laid upon the exposure is sealed in the cavity by means of softened temporary stopping applied without pressure.

If the cavity be inaccessible, where the rubber-dam cannot be used to exclude fluids, and where an arsenical application cannot be made with precision and without fear of dislodging it while it is being sealed in, it is advisable to form a special cavity for its reception. This should always be made when possible in a line of direct approach to the pulp-canals. The cavity is to be made as deep as possible without plunging into the pulp. As a rule, in the conditions demanding extirpation of the pulp the dentin is insensitive or nearly so, so that the pulp may be almost exposed without pain. An exception to this, however, is found when pulp-nodules exist, when the dentin may be exquisitely sensitive. In these cases two or more applications of the paste are required; as soon as the pulp can be exposed, a direct application of the paste is to be made.

To make the application, the cavity is sterilized, placed under rubber-dam, and an application of pyrozone is made, remaining five minutes or longer; the cavity is dried, and a piece of cotton, not larger than a pin-head, is to have a minute portion of the paste placed upon it, and then laid gently upon the spot of exposure. It is the general practice to seal in the arsenic with cotton and sandarac, or temporary stopping. The first becomes very foul in twenty-four or forty-eight hours, and swells, causing pressure upon the pulp and much pain. Temporary stopping, made very soft, may be manipulated so that it causes no pressure, but some slight pressure is the rule; so that a thin paste of zinc phosphate is to be recommended as the sealing-material. "Flowed over the arsenical application and removing the rubber-dam before the cement has hardened will diminish or prevent the pain incident to the application and render the cement easy of removal."[1]

[1] Miller.

Instead of placing the paste upon a pledget of cotton, cotton-fibre may be rolled in an arsenical paste, dried, and preserved; in this condition it is known as devitalizing fibre. The advantage of this fibre is that a dry arsenical application may be made to the pulp, so that there will be no danger of oozing. The length of time required for devitalization is usually about twenty-four hours, although in a large number of cases sensitivity will still exist at the upper third of the canal portion of the pulp at the end of two days. At the end of four or five days the pulp will in most cases have completely sloughed at the apex. To relieve unusual pain following an arsenical application general anodynes or sedatives will at times be required. While morphia sulfate, gr. $\frac{1}{6}$, will deaden the pain, the after-effects of morphia are uncomfortable. The coal-tar derivatives are useful; gr. x of ammonol, or phenacetin and exalgin, āā. gr. iij, administered at the time of the application and repeated upon retiring, will act effectively.

An annoying type of case is where approach is to be made in an approximal cavity with a cervical margin beyond the gum-margin, where access to the pulp-canals will be direct after devitalization, and yet the cavity cannot be kept dry. Such cases are managed after the following manner: a small piece of temporary stopping is softened and pressed against the cervical wall of the cavity, but not covering the pulp-exposure; the stopping is then moulded against the gum pressing it back (Fig. 293): this guard accurately placed will protect the gum against arsenical poison by preventing oozing of the latter from about the cervical edge. An application of devitalizing fibre is then made, and sealed as usual.

Fig. 293.

Cervical cavities not having a retentive form, and abraded teeth, offer difficulties, which are overcome by drilling a special pit for the reception of the paste.

In cases where there are pulp-nodules, and where chronic degenerations of the pulp exist, the arsenic is removed at the end of the usual devitalizing period and free entrance is made to the pulp, cutting away all insensitive portions; if pulp-nodules can be lifted away painlessly, they are removed, and a fresh application of arsenic is made, to remain again several days. In all of these cases, to effectually devitalize it may be necessary to apply a paste of arsenic trioxid in glycerin, or in one of the essential oils.[1]

Removal of Pulp.—At the end of four or five days the dressing seal, and cotton containing the paste are removed, the cavity freely syringed with hydrogen dioxid, and the rubber-dam applied. Large

[1] Miller.

sterilized rose burs are used to open the pulp-chamber freely and to remove *all* softened dentin (all softened dentin being *always* removed from pulpless teeth).

The cavity is now to be given such form that pulp-broaches may be passed directly and freely to the apex of each root. This rule is to be followed, no matter how much tooth-substance is sacrificed to carry it into effect. As the future health of the tooth depends almost entirely upon the thoroughness with which each canal is cleansed, sterilized, and hermetically sealed at the apex, it is evident that the removal of crown-tissue is a small evil compared with incomplete entrance to and cleansing of a canal.

A new and perfect pulp-broach is dipped in carbolic acid and gently passed to the apex of the root; the teeth of the broach are turned away from the pulp until the instrument is fully inserted, when the broach is turned so that its teeth shall engage the entire length of the pulp, which may then usually be removed entire. In multirooted teeth, after removing the body of the pulp, the largest canal is first entered and the pulp removed; this canal is then to be loosely filled with a twist of cotton containing an antiseptic; carbolic acid, formalin in 2 per cent. solution, or hydronaphthol. The next largest canal is cleansed and treated in the same manner, and after this the smallest canal.

Removal of the pulp entire may nearly always be assured if, after an arsenical application have been in the tooth for three or four days, it be removed, and a dressing of formalin, 5 per cent., be inserted for several days. The pulp is rendered tougher by this agent.

In canals too small to admit the pulp-broaches, cleansing and uniform enlarging of the canals are accomplished by means of chemical agents. The general cavity-wall is varnished to prevent the action of the acid upon the dentin, and by means of a pair of Flagg's dressing-pliers or a minim-dropper a drop of sulfuric acid (50 per cent. solution) is deposited at the mouth of the canal to be operated upon. The finest size of Donaldson's canal-cleanser is then passed into the canal as far as it will go, using a pumping movement to carry the acid further into the canal and to scrape the canal-walls softened by the action of the acid.[1] The acid chemically destroys any organic matter—*i. e.*, pulp-tissue—present, releases the calcium of the dentin from its combination, and forms calcium sulfate, which is mechanically removed by scrapers. The operation is continued until the apex of the root is reached.

If all of the operations have been carried out with antiseptic precautions, sterilizing the cavity, placing it under rubber-dam, drenching the pulp with antiseptics, and using none but sterilized instruments, the canals are now in an aseptic condition and are to be hermetically

[1] Callahan, *Proc. Ohio State Dental Society*, 1894.

sealed—filled. If the pulp have been removed within two days from the application of the arsenic, bleeding may follow the extirpation of the pulp, or a mild and what should always be a transient pericementitis may arise. In case of bleeding, applications of hydrogen dioxid will act as a styptic and also decompose and remove the blood. The canal should not be sealed until the bleeding ceases. The transient pericementitis is most common in cases where slight sensitivity of the apical portion of the pulp existed at the time of extirpation, and is evidenced by soreness of the tooth, tenderness upon percussion. This irritation may be pronounced if immediate root-filling, the usual procedure in cases of intentional devitalization and removal of the pulp, be practised. If tenderness appear during or immediately after extirpation, it promptly subsides upon filling the pulp-canals with a saturated solution of menthol in chloroform, filling the canals loosely with cotton, hermetically sealing the cavity, and painting the gum overlying the tooth with tr. iodin. The canals should remain unfilled for a week, until all evidences of pericemental irritation subside. Before filling, the canals should be thoroughly washed with hydrogen dioxid.

The Root Canal-filling.—The features to be possessed by a canal-filling should be: first, it should be non-irritating; secondly, it shall hermetically seal the canal; thirdly, it shall be unalterable in the conditions surrounding it. If possible, it should be continuously antiseptic, and be removable if subsequent conditions ever demand its removal. The materials most employed are zinc oxychlorid, and gutta-percha, solid and in solution, or combinations of both.[1]

The present tendency is to substitute melted paraffin, containing antiseptics, for other materials. The choice is clearly indicated as a rational therapeutic measure. Certainly it will be evident from a study of the forms and variations of pulp-canals (see Chapter VIII.) that only pastes or fluids can fill perfectly all of the irregular spaces found in pulp-canals.

Paraffin fulfils all of the conditions required of a correct canal-filling. In using this material a small piece is caught between the jaws of a pair of Flagg's dressing-pliers and held over a flame until melted; then the closed beaks are placed as high up the canal as they will go, and slowly withdrawn; gradually opening the beaks, the fluid runs up the canals of even upper teeth. A warm, smooth probe is then used to pump the fluid paraffin into all parts of the canal. Into the fluid paraffin a long slender metallic point of some unoxidizable material—gold, platinum, or aluminum—is warmed and thrust. The end of the metallic point is left projecting into the pulp-chamber, so that should removal of the canal-filling ever become necessary, a hot instru-

[1] See *American Text-book of Operative Dentistry.*

ment may be laid against its exposed end, melting the paraffin, when the point may be withdrawn.

Accidents with Arsenic.—If arsenic trioxid come in contact with any vital tissue, it exercises its destructive influence upon it. The most common accident is the oozing of arsenic from beneath a sealing application, and its contact with gum-tissue. The effect of the arsenic in these cases depends upon the point of lodgement. Arsenic trioxid being insoluble in the fluids of the mouth, may remain lodged in a minute crypt and exercise its destructive influence. Accidents in this direction should be guarded against by carefully pressing away the gum from cavities prior to using the arsenic; by using a very minute portion of the latter and placing it with precision; by the use of small pieces of devitalizing fibre instead of paste. Oozing, however, should never occur, and when it does, it is evidence of lack of care, or, worse, of gross carelessness upon the part of the operator.

Arsenical applications sealed in with any material other than zinc phosphate should be examined at the end of twenty-four hours to see that there is no evidence of irritant poisoning of the soft tissues. The evidences of the presence of arsenic in contact with gum-tissue are: deep purple engorgement of the gum and subsequent sloughing of the poisoned tissue. If a portion of the arsenic gain lodgement far beneath the gum-margin, near the pericementum, it may exercise its necrotic effects upon the latter tissue, destroying it in part or in whole. Flagg has recorded cases in which necrosis of the contiguous alveolar process has occurred.

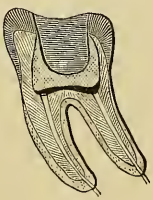
The only cure of the condition consists in the thorough removal of every particle of the arsenic. Being insoluble, it must either be washed away mechanically or be transformed into a soluble or an inert body. The swollen gum-tissue is to be pressed away from the tooth and jets of warm water thrown forcibly into the space in the hope of dislodging and washing away the insoluble arsenic. A freshly prepared mass of ferric hydrate (the antidote of arsenic), made by adding magnesium oxid to ferric chlorid, may be packed into the pocket, but its utility is questionable. The free use of iodin tincture in one case appeared to give good results, perhaps from the formation and washing away of arsenic iodid. Any projecting masses of œdematous gum should be cut away, as they are dead and will slough at any rate, and a freer access to deep parts is had—the blood-flow may itself wash away the arsenic. The forcible washing should be prolonged and repeated. The tissue to whatever extent it has been devitalized by the arsenic will slough away. Dental literature contains the records of a few cases in which the action of arsenic has extended beyond the ends of the roots of teeth, when an arsenical application has been made far up a canal to destroy

a vital pulp-filament. One case is recorded where an application was carried bodily beyond the apex of the root. The records of these cases are not sufficiently clear to formulate rules as to the extent of destructive action caused by minute portions of arsenic. *A priori*, they would be governed by the amount of arsenic which gains access to the pericementum; certainly more or less alveolar necrosis would be the natural result. The insertion of a broach into and beyond an arsenical application high up in the root, into the apical foramen or beyond, may no doubt account for rare cases of uncontrollable pericementitis, ceasing only with the loss of the tooth, and, it may be, of contiguous bone.

PARTIAL REMOVAL OF PULP.

The cobalt method of pulp-treatment has been alluded to. Wm. Herbst, of Bremen, advanced the idea that if the bulbous portion of the pulp be devitalized by cobalt and removed, leaving the root-portions, the latter will remain vital, if protected after a manner described by him. The bulbous portion of the pulp is cut away and the pulp-chamber enlarged by means of large rose burs. Over the pulp-stumps a cylinder of tin-foil is laid, and burnished to fit the floor of the pulp-chamber, without pressure upon the pulp-stumps (Fig. 294). Over this a filling is placed. Herbst claims, endorsed by Bödecker,[1] that the pulp-stumps will remain vital. Were this to be depended upon, it would be a marked saving of time and trouble, and would lessen the chances of pericementitis subsequent to pulp-removal; but when it is known that the cobalt of Herbst is metallic arsenic, the ultimate death and decomposition of the pulp-remnants seem almost inevitable.

Fig. 294.



MUMMIFICATION OF THE PULP.

As early as the introduction of arsenious oxid as a devitalizing agent it was noted that a certain percentage—or, rather, an uncertain percentage—of cases gave evidence of little or no disease after the application of arsenic and its sealing in a cavity by filling. Later, it was found that applications of powerful antiseptics to exposed pulps not infrequently were followed by a long-continued quiet of that organ; still later, when more definite knowledge was possessed of the pathological results which might follow the leaving of portions of pulp-substance in the canals of teeth after devitalization by arsenic, it was observed that after saturating the canals with creosote or zinc-chlorid solutions,

[1] *Anatomy and Pathology of the Teeth.*

many cases gave little or no evidence of pericemental disturbance thereafter.

While it is unquestionably preferable to always thoroughly remove the last vestige of devitalized pulps, the time, care, skill, and expense involved in perfect cleansing are drawbacks to its universal practice. The only other possible solution of the difficulty is to so alter the tissue not removed that it shall remain permanently aseptic, and, if possible to make it so, antiseptic.

Observations derived from clinical experience although undoubtedly of great and permanent value, are indeterminate, and our truly scientific knowledge of this matter dates from W. D. Miller's experiments.[1] He credits Witzel with the first systematic observations in this direction. Witzel, in 1874, "devitalized the crown-portion of pulps by means of arsenic, extirpated that portion, leaving the pulp in the canals undisturbed, their exposed ends being treated as freshly exposed pulps." This is the method followed by Herbst, who employs cobalt (which is native arsenic sulfid or metallic arsenic) instead of arsenic trioxid.

Miller's experiments have shown that none but the most powerful and penetrating antiseptics have value as permanent sterilizers. These are the cyanid, bichlorid, and salicylate of mercury, sulfate of copper, and oil of cinnamon. Orthocresol, carbolic acid, trichlor-phenol, and zinc chlorid penetrate the pulp-tissue rapidly, but are too diffusible, disappearing in a few weeks.

He classifies salicylic acid, eugenol, campho-phénique, hydronaphthol, α- and β-naphthol, acetico-tartrate of aluminum, and some essential oils, resorcin, thallin, sulfocarbolate of zinc, etc., as being of doubtful value.

Those nearly or quite worthless are iodoform, basic anilin coloring-matters, borax, boric acid, dermatol, europhen, calcium chlorid, hydrogen dioxid, sozoiodol salts, tincture of iodin, spirit of camphor, and naphthalin.

The preparation giving the best results consisted of mercuric chlorid, 0.0075 gram; thymol, 0.0075 gram, in tablet-form.

The pulp is devitalized; the crown-portion and all the root-portion readily accessible are removed; one of the tablets is placed in the pulp-chamber, crushed by means of an amalgam-plugger, and covered with gold-foil. The mercury salt tends to discolor the crown of the tooth, so that its employment should be restricted to the posterior teeth; indeed, the necessity for its use would be, as a rule, found with these teeth, being those from which it is most difficult to extract pulp-remnants. Miller expresses faith in the power of oil of cinnamon to permanently sterilize pulp-fragments. He suggests the experimental

[1] *Proc. Columbian Dental Congress*, 1893.

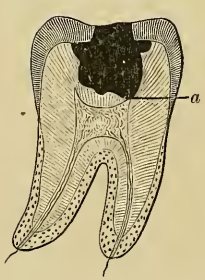
application of the sterilizing tablets to such teeth as are readily salvable, yet which are for various reasons "consigned to the forceps."

Theodore Söderberg, of Sydney, N. S. W., reports excellent results from a continuous practice of this method of pulp-sterilization. He employs a paste composed of—

℞. Alum exsic., Thymol, Glycerol, āā. ʒj;
Zinc. oxid., q. s. to make stiff paste.—M.

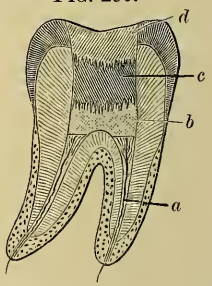
He substitutes dried alum for tannin, originally used by him as the hardening-agent; his experiments showed the tannin to be productive of discoloration. Mercuric chlorid is set aside for the same reason. Oil of cassia employed in the paste also caused discoloration. At present Söderberg adds a small quantity of cocain to the paste to prevent the pain arising from the action of the dried alum. He states (Nov. 1895) that he has in a year applied the paste in 97 cases, and has had no untoward results. The method of placing the material is shown in Figs. 295 and 296.

Fig. 295.



a, caries exposing a horn of the pulp.

Fig. 296.



a, root-portion of pulp; *b*, mummifying paste; *c*, zinc phosphate; *d*, gold or amalgam.

C. A. Firth, of Queenleyan, N. S. W.,[1] advises the omission of zinc oxid from the paste, to avoid the formation of the brown tannate of zinc. He suggests the use of a mixture of tannic acid and thymol, equal parts, made into a paste with glycerol, and applied with *ivory* instruments, to avoid discolorations. He expresses himself as gratified at the results obtained.

[1] *Dental Cosmos*, May, 1896.

CHAPTER XX.

GANGRENE OF THE PULP.

Definition.—By gangrene of the pulp is meant its death *in toto* from being cut off from its nutritive supply at the apex of the root. It occurs in two forms, dry and moist. Dry gangrene is the condition known as mummification of the pulp. Moist gangrene is associated with putrefactive decomposition of the organ. The conditions differ not only as to causation, pathology, and morbid anatomy, but in their effects and treatment, so that each requires separate consideration.

Dry Gangrene of the Pulp.

Definition.—By dry gangrene of the dental pulp is meant its death *in toto* and its subsequent transformation into a dry, shrivelled mass occupying the pulp-chamber and canal.

Causes.—The causes of this condition may be divided into physical and chemical. The physical causes, so far as known, are direct injury or jugulation of the vessels of the pulp at the apical foramen. This may be due to blows upon the teeth, to their rapid movement in regulating, particularly by their forcible movement by forceps, to too sudden and violent wedging, and by non-fixation of the teeth during and subsequent to regulating, permitting their undue movement; by any force, in short, which can cause torsion or tension of the vessels at the apex of the root. These influences acting upon the apical vessels may cause strangulation, and if the access of air and organisms be impossible or does not occur, the watery parts of the pulp may be removed, leaving the organ as a tough, shrivelled mass.

The chemical causes include the action upon the pulp of agents which have either the power to devitalize it and preserve it, or which transform it into a dry, shrivelled, and aseptic mass after its death.

Nearly all recorded cases appear to have followed the employment of zinc oxychlorid as a pulp-capping or a cavity-lining.

Pathology and Morbid Anatomy.—Upon opening a pulp-chamber containing a mummified pulp no odor is emitted, and the pulp is seen dark, dry, and shrivelled. In cases of pulp-death under oxychlorid caps it is difficult or impossible to determine whether the pulp has been killed by the zinc chlorid and preserved by absorption of that substance, or whether it has died from other causes and the zinc salt

has acted as a preservative. Certainly the condition does not appear to be recorded in connection with other capping-materials, although other antiseptics, such as those described under the head of mummifying pastes, might undoubtedly produce similar conditions.

Symptoms.—Unless secondary processes arise, mummified pulps give rise to no symptoms, and the existence of the condition is usually discovered by accident. Their usual history is as follows: at a previous time (perhaps years before), an exposed or almost exposed pulp has been covered with a cap or cavity-lining of the oxychlorid of zinc, and remained comfortable thereafter. At some subsequent time it may be necessary to open the tooth, usually on account of recurring caries: the total absence of dentinal sensitivity is noted; the tooth has changed color but little, if at all; and the operator burs carefully toward the pulp to determine its condition. (It should be remarked here that absence of dentinal sensitivity in a tooth having normal color and which contains a very large filling is an indication of aseptic death of the pulp, and the operator should renew all of his antiseptic precautions as to isolation of the tooth by the rubber-dam and complete sterilization of all instruments, and of the territory of operation.) The burring is continued without any evidence of sensitivity, and the instrument is finally felt to pass into the pulp-chamber. There is no odor, no escape of fluid; the pulp is found dry and shrivelled. If sterilized pulp-extractors are passed into the canals, the remnants of the pulp may be withdrawn, exhibiting none of the usual signs of decomposition, such as odor and confluent softening.

Treatment.—The treatment consists in aseptic cleansing and hermetical sealing of the pulp-canal. If the fluids of the mouth be permitted access to the mummified pulp, infection occurs promptly, and a vigorous pericementitis may be lighted up. Whether the dried pulp affords a favorable breeding-ground for particularly virulent organisms, or whether the apical pericementum in such cases affords a most suitable field for their secondary activity, certainly the pericementitis which sometimes follows the septic opening of such cases is peculiarly obstinate. In any tooth which has not suffered any change of color, in which dentinal sensitivity is entirely absent, and which was believed to contain a vital pulp, precautions as to asepsis and antisepsis should be redoubled. The general rule, that no pulp-chamber should be deliberately opened before adjusting the rubber-dam, has here an increased significance. Every bur that is used should be thoroughly sterilized before using. As soon as the pulp-chamber is opened, that cavity is explored. A clean, new broach which has been dipped in carbolic acid is carried to the apex of the root and withdrawn; if the pulp be mummified, the broach brings it away and exhibits no fluid upon it. If any deposits

are found on the broach, it is evidence that some decomposition has occurred. The agent indicated in that event is one which will effectually destroy all organisms which might be present and which will chemically decompose—transform into soluble and entirely removable substances—the products of decomposition. Sodium dioxid is, therefore, the indication. It may be used in 50 per cent. solution pumped into the canals by means of aluminum or iridium broaches. After several applications of the sodium dioxid solution have been made, the canals are to be washed out with a 10 per cent. solution of hydrochloric or sulfuric acid and dried.

No matter what precautions are taken, the possibility of infection must always be borne in mind. While pericementitis usually appears promptly in these cases, if infection have occurred, it may not arise for two or more days; during this period it is advisable to insert a probationary filling, one which can readily be removed if pericementitis does arise. This filling may be a mixture of salol and aristol, paraffin and aristol, or a twist of cotton saturated with one of the antiseptic oils, such as cassia oil. In all cases where incomplete mummification of the pulp is suspected, it is a wise precaution to seal in the cavity a 5 per cent. solution of formalin for a day or two before broaching, to give assurance of complete sterilization.

Moist Gangrene of the Pulp.

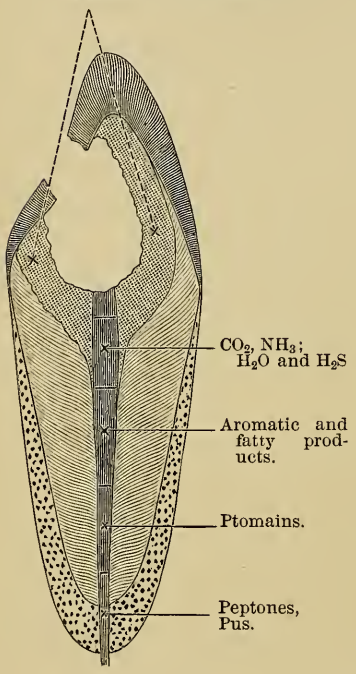
Definition.—By moist gangrene of the pulp is meant, the death of the pulp *en masse* and its subsequent decomposition by the action of putrefactive agencies. As putrefactive decomposition is the essential feature in these cases, and that which gives the process its pathological significance, the causes, nature, effects, and treatment of putrefactive decomposition of the pulp are included under this sub-heading.

Causes.—The death of pulp-tissue from any cause and its infection with the bacteria of putrefaction. The cases are of two types—those in which decomposition occurs without the existence of an opening communicating with the exterior and those in which such an opening exists. The first class may be subdivided into those in which decomposition occurs subsequent to the insertion of a filling which hermetically seals the space between the pulp and the exterior, and those in which the tooth is non-carious. It is to be recognized that in the absence of bacteria putrefaction cannot exist. By putrefaction is meant that serial, progressive decomposition through which albuminous substances are finally resolved into the end-products, hydrogen sulfid (H_2S), carbon dioxid (CO_2), ammonia (NH_3), water (H_2O), and hydrogen phosphid (PH_3). One distinguishing feature of the process is the evolution of malodorous gases.

Cases of Open Cavities.

When the pulp of a tooth is exposed and becomes the seat of that series of vascular and nutritive disturbances—hyperæmia, inflammation, and suppuration—eventuating in its gradual death, the necrotic portions undergo putrefactive decomposition. Several processes are in operation at the same time, so that different portions of the pulp exhibit differences in chemical composition, differences in the nature of the infection, and also in the pathological conditions existing. For example, while the apical portion of the pulp is the seat of inflammation and suppuration, the portion of the pulp previously destroyed through these processes, is the seat of later stages of chemical destruction, until that portion which was first acted upon is being resolved into the end-products of albuminous decomposition, of putrefaction (Fig. 297). In this serial decomposition albuminous substances are first transformed into peptones and allied substances, some of them being very toxic. Compound ammonias, known as ptomaïns, or animal alkaloids, are probably next formed. Next the nitrogenous bases—leucin, tyrosin and the amins (methyl, ethyl, and propyl)—make their appearance together with organic fatty acids. Next aromatic products, indol, phénol, cresol, etc., and finally hydrogen sulfid, ammonia, carbon dioxid, and water. By alternating processes of hydration, reduction, and oxidation, bodies of increasing simplicity of chemical composition are formed.[1]

Fig. 297.



Miller[2] found in the deepest portions of the degenerating, putrefying pulps, where inflammation and suppuration were in progress, a preponderance of small cocci and diplococci, and proceeding toward the open pulp-chamber an increasing number of large cocci, several forms of bacilli, vibrios, and other spirillæ, spirochætæ, and long thread-forms (Figs. 298–305). Figs. 304 and 305 are from the same pulp; Fig. 305 was taken from the radicular portion of a pulp which was alive and suppurating; Fig. 304 was from the putrid crown-portion.

[1] Ziegler's *General Pathology.*

[2] *Dental Cosmos,* 1894.

Until infection of the pericementum occurs these cases give rise to no symptoms, except odor.

FIG. 298.

FIG. 299.

FIG. 300.

FIG. 301.

FIG. 302.

FIG. 303.

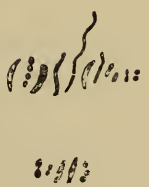


FIG. 304.

FIG. 305.

CASES OF PUTREFACTION UNDER FILLINGS.

When a filling is placed over an infected pulp, or when the pulp dies subsequent to the insertion of a filling, the organ undergoes decomposition, the decomposition being carried on in this instance without the access of air—*i. e.*, is accomplished by anaërobic organisms. Miller found that bacteria of pulp-putrefaction cultivated in gelatin, with and without the access of air, exhibited a difference in

the poisonous properties of their products. Those developed with free access of air produced stronger reaction, and more extensive suppuration than those developed without the access of air.

Symptoms.—Prior to infection of the pericementum, pulp-putrefaction occurring under fillings may give rise to much pain. First, in chronic abscess of the pulp, by imprisonment of the pus. Heavy, throbbing pain, indefinitely located, may be felt, and the tooth may be tender upon percussion (see Suppuration of Pulp). Applications of heat are almost invariably followed by a paroxysm of pain; the gases of putrefaction being expanded by heat, exercise pressure upon the still vital portions of the pulp and upon the pericementum; as regards the latter tissue, however, it is probable that poisonous products are by the expansion of gases forced into it. The relief from pain is in some cases almost instantaneous when an opening is made into the pulp-chamber, giving vent to the imprisoned gases.

Cases of Pulp-putrefaction without Previous Caries.

Under the head of dry gangrene were described several varieties of traumatism which caused the death of a tooth-pulp *en masse*, particularly in single-rooted teeth. Instead of becoming mummified, the necrotic pulp may undergo putrefactive decomposition. It can only be a matter of conjecture how the organisms necessary to putrefactive decomposition gain access to the pulp. The possibility that organisms may make their way through the cementum and dentin at the neck of the tooth must be admitted, although its extreme improbability must be recognized. The more probable explanation of their presence is that they have gained access to the circulation from some other portion of the body, and have been deposited in the dead pulp, and, finding there a suitable soil, have developed.

In one class of cases the source of infection is evident. In cases of phagedenic pericementitis (which see) the degeneration of the pericementum may open a direct passageway from the mouth to the tissues of the apical space; the pulp is cut off from its vascular and neural connections, dies, and undergoes putrefaction. This condition may be noted upon upper molars particularly, where the pericemental degeneration has involved the apical tissues about the palatal root of the tooth; upon opening the pulp-chamber the pulp is found dead and putrefying in whole or in part.

Many of these cases offer no subjective evidences of disturbance, attention being directed to the tooth by a growing opacity of the dentin. A reaction occurs between the hydrogen sulfid and decomposing hæmoglobin (hæmosiderin) of the red blood-corpuscles, forming dark compounds, which infiltrate and discolor the dentin, rendering it opaque.

In other cases infection of the pericementum may occur, leading to chronic pus-formation, with an exit near, or in many cases far distant from the tooth.

A confusing condition clinically is found where one-half of a pulp has died and undergone decomposition, as in lower molars, the other half remaining vital, although the seat of infection and inflammatory action. So far may this condition go, that abscess, acute or chronic, may be present upon the root of one tooth long before the second segment of the pulp has succumbed. The diagnosis of such cases is made by obtaining the painful reaction to heat, and usually some tenderness upon percussion upon some particular portion of the tooth; upon opening the tooth the peculiar condition described is formed.

Treatment.—The general principle of treatment is the same in all of these cases, no matter to what clinical division they may belong—the disinfection of the canals, the removal of all decomposed and decomposing pulp-tissue, prevention of infection of the pericementum, and hermetical sealing of the apex of the canal.

In all of the cases the imminent danger, and that to be guarded against, is to avoid mechanically carrying a portion, ever so minute, of infective material past the apical foramen. There is but one way to accomplish this end with certainty, and that is to render the contents of the canal absolutely non-infective before mechanical procedures are instituted. This implies the use of an antiseptic which will gradually diffuse through the putrefying mass to the apex of the root, and one which is certainly germicidal. The condition, as shown, is one of infection to near the apex, so that any pressure exerted upon the putrid mass may force organisms or their waste-products through the apex of the root. Many antiseptics have been shown to have the desired power; notably the essential oils of thyme and cinnamon; carbolic acid and other coagulating agents are less diffusible, hence less prompt and certain in action; lysol, an allied substance, is, however, much more diffusible. Of all present antiseptics solutions of formalin are perhaps most effective because most diffusible.

The first step of operative procedures is gaining access to the putrid pulp. As a preliminary, the mouth is washed with antiseptics—hydrogen dioxid, potassium permanganate, or meditrina—and the rubber-dam adjusted. The tooth is next drenched with the same antiseptic and partially dried. In cases of open cavity loose débris is lifted away, without exercising pressure, until full exposure of the pulp-chamber is secured. A pellet of cotton saturated with meditrinia (full strength) laid gently upon the mass deodorizes it.

Cases of putrid pulp under fillings, as a rule, are only seen when evidences of pericemental disturbance have appeared. Fissure-burs

are passed around the margins of the filling until it is loosened and detached, and free applications of the antiseptic wash made until free access to the pulp-chamber is had.

In cases without cavities or fillings a spear-pointed drill is passed by the most direct route to the axis of the pulp-canal. The drill is to be advanced gently, so that it will not plunge forcibly into the pulp-chamber. In all of these cases, as soon as free access to the pulp-chamber is secured, a drop of a 10 per cent. solution of formalin is flowed into the cavity and a pellet of cotton wet with the same solution is gently laid over it, which is to be sealed in for twenty-four hours by means of temporary stopping.

It is a general clinical experience that in the cases of putrefying pulp in non-carious teeth, pericementitis is a frequent sequel to the opening of the pulp-chamber. This has been attributed to the entrance of organisms by way of the artificial opening made. Other reasons for infection would be the access of air to the previously closed cavity, favoring the development of virulent organisms already present, but inactive on account of an absence of free oxygen; or, again, the mechanical forcing of infective material beyond the end of the root. The first cause is scarcely probable; the second more probable; and the third the most probable cause of the infection. Presumptive evidence of this is found where, as advised, an application of formalin is placed in the tooth for twenty-four hours before any attempt is made at the mechanical cleansing of the canals; subsequent pericementitis rarely occurs.

To remove the contents of the canals after sterilization no agents act with such promptness and effectiveness as solutions of sodium dioxid, or of Schreier's alloy of sodium and potassium—kalium-natrium. The reason for this is clearly seen from a study of the nature of the substances contained in the canals, and the reaction which occurs when the alkalies mentioned are brought in contact with them. Brought into contact with agents containing water, potassium-sodium immediately causes its decomposition, abstracting HO, forming sodium and potassium hydrates; hydrogen is set free, which ignites in consequence of the heat of chemical combination set free in the union of the metals with hydroxyl. Sodium dioxid brought in contact with organic matter gives up its extra atom of oxygen to the latter and is reduced to N_2O, sodium oxid, which, combining with water, becomes sodium hydroxid. Sodium hydroxid from either source saponifies all fatty matters and dissolves albumin and its derivatives. The action of sodium-potassium is very pronounced; a minute portion of the alloy being brought into contact with decomposing organic matter, decomposes its water with such activity that a

spark is produced. The germicidal action of the material has been attributed to this heat; the correct explanation is probably the activity of sodium and potassium hydrates in their freshly formed state. The extra atom of oxygen in sodium dioxid acts as a prompt antiseptic; and the active sodium hydroxid formed fulfils its function as a saponifying and solvent agent. Sodium dioxid may be used either in dry powder or in saturated solution for the purpose named.

Non-oxidizable metals are to be preferred in making applications of these agents; broaches of aluminum or of iridio-platinum answer the purpose. If the material be used dry, either sodium-potassium or sodium dioxid, the roughened broach is dipped in the chemical agent and passed part way up the canal of the tooth or up the largest canal in multirooted teeth; a vigorous reaction immediately occurs between the chemical agent and the canal-contents; as soon as this ceases the canal is wiped out with a wisp of cotton and a deeper application made. The alternate application and wiping away are continued until the apex of the root is reached. By this time the walls of the canal are seen to be distinctly bleached by the action of the oxygen set free. It is always to be remembered that this decomposition represents a chemical reaction in which there is a distinct quantitative relation between the amount of decomposer and decomposible matter: an excess of the decomposer is desirable or even essential. In the vast majority of cases an error is made the other way. The operation of thoroughly decomposing the contents of a pulp-canal and dentinal tubuli containing decomposing albuminous matter, requires a considerable length of time, as will be seen in the following test. After having spent a half an hour or longer in carrying successive portions of the active agents named into pulp-canals, say of a lower molar, until all evidences of chemical reaction cease, then forcibly syringe the canals with hydrogen dioxid or a 10 per cent. hydrochloric acid solution until effervescence ceases; dry the cavity and canals, insert a ball of cotton in the pulp-chamber, leave the canals unfilled, and seal the crown-cavity for several days; at the expiration of this time unseal the tooth, remove the cotton, and in a number of cases the odor of putrefaction may be detected. The sodium oxid solution slowly makes its way into the dentinal tubuli, decomposing their contents. If, now, a 10 per cent. solution of sulfuric acid be pumped into the canals, it effects the decomposition of the sodium compounds present, forming with Na_2O_2 [1]

$$Na_2O_2 + H_2SO_4 = Na_2SO_4 + H_2O_2,$$

solutions of sodium sulfate and hydrogen dioxid, which in its turn is

[1] See Kirk, *American Text-book of Operative Dentistry.*

decomposed into water and oxygen, the latter driving out the altered contents of the tubuli. Any sodium oxid present is transformed by the sulfuric acid into sodium sulfate and water. The evidence of thorough action of the sodium dioxid is the bleaching of the dentin; dentinal walls unbleached are evidence of incomplete action of the sodium compound.

It has been advised by many operators that such canals be immediately and permanently filled, as, indeed, they may be in very many cases, and no subsequent trouble arise. It is the part of prudence, however, to fill the canals temporarily until it is seen that no infection of the pericementum has occurred. During the period of probation, the canals are to be filled with a diffusible antiseptic; oil of cassia is the agent most frequently and acceptably used for this purpose. Salol makes an excellent tentative filling in such cases; it is combined with one-third its volume of aristol for this purpose. It is melted and flowed into the canals by means of Flagg's dressing-pliers, and while fluid a hot cone of metal is thrust into the fluid mass. The canal-filling may be made of cotton-thread dipped in an antiseptic oil, if preferred.

In a few days, or, better, a week, if no evidences of pericemental disturbance appear, in excess of a slight and transient soreness, the tooth may be opened, always under rubber-dam, the canals cleansed, dried, and permanently filled. It is always a wise precaution to place 25 per cent. pyrozone in such canals for five minutes or longer before drying and filling the canals. Slight pericementitis, evidenced by tenderness of the tooth upon percussion, may immediately follow the treatment of canals by the sodium compounds, caused by the passage of a minute portion of the preparation beyond the apical foramen. As a rule, the irritation is but transient, and is soon reduced by applications of a counter-irritant upon the gum of the affected tooth—tr. iodin, tr. aconite, and chloroform, in equal parts, painted on the gum. More severe reactions indicate active pericementitis.

Canals containing putrescible material which are too fine to admit even slender broaches are, after the action of the formalin solution, entered and cleansed by means of the sulfuric acid method. Enlargement of the canals and destruction of the putrefactive matter are accomplished simultaneously.

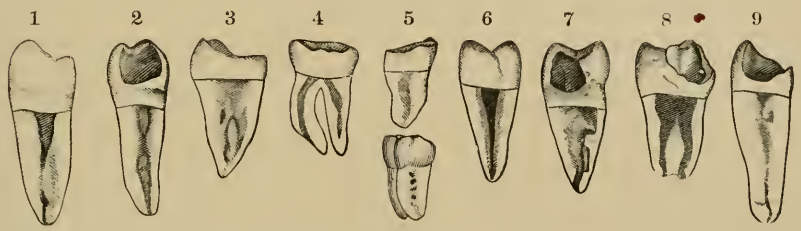
It has been maintained that if the pulp-canals could be lined with, and the contents of the dentinal tubuli be transformed into, permanently antiseptic material, that future sepsis would be rendered impossible.[1] L. P. Bethel,[2] basing his procedures upon the fact that dentin impregnated

[1] *Proc. American Dental Association*, 1896.

[2] *Proc. New York Institute of Stomatology*, 1897.

with silver nitrate notably resisted or prevented the progress of dental caries, conceived that if the same agent could be made to permeate the dentin of tooth-roots, it would act there as a permanent antiseptic and prevent future putrefaction and bacterial development. The tooth is isolated; the application is confined to posterior teeth; the danger of dentinal discoloration through the reduction of the silver salt is too great in the anterior teeth, and is only designed for canals of such size and shape that mechanical cleansing and filling are extremely difficult. The crown-cavity is to have its walls covered with wax or varnish to prevent the passage of the silver nitrate into the crown-dentin; the canal is pumped full of a silver-nitrate solution (25 per cent. to 75 per cent.), a pellet of cotton containing the same solution is wrapped around the positive electrode of a cataphoresis apparatus, the current is applied, and the silver solution is driven into all of the tortuosities of the canal (Fig. 306). The silver combines with the contents of the dentinal tub-

FIG. 306.



1. Operated on in the mouth with a 50 per cent. solution silver nitrate. Crown-cavity protected from discoloration by a thin coating of melted wax.
2. Operated on in the mouth with a 75 per cent. solution silver nitrate. Crown-cavity protected with wax.
3. Operated on in the mouth with 75 per cent. solution silver nitrate.
4. Shows perfect lining formed, and penetration of the silver nitrate into the dentinal tubuli.
5. Freshly extracted tooth operated on outside the mouth. The crown and roots were filled with decomposing material which was not removed, the electrode and nitrate being applied to the surface; still the nitrate permeated the canals. Exposed surfaces of both canals shown.
6. Operated on outside of mouth. Foramen on inside of root.
7. Shows penetration in flat root with restricted and branching root-canal. Could not get broach more than one-eighth inch into canal.
8. Operated on outside of mouth, for twelve minutes, attempting to force the silver nitrate through foramen of root.
9. Shows returning branch of canal that might easily be left unfilled.

uli, forming silver albuminate; the nitric acid is formed at the positive pole (the electrode), giving an acid reaction to the canal-contents; the acid is neutralized with ammonia. Unless a very high voltage be applied, the silver does not penetrate the dentin to any considerable depth and it is not desired to have it do so. Crede's experiments indicate that metallic silver acts as an antiseptic by being oxidized by bacterial products, the argentic oxid being afterward transformed into antiseptic salts of silver by bacterial waste-products, notably by lactic acid, silver lactate being formed.

SECTION V.

DISEASES OF THE PERICEMENTUM.

CHAPTER XXI.

SEPTIC APICAL PERICEMENTITIS (ACUTE).

Classification.—The dental periosteum and ligament, or the pericementum, is the seat of numerous nutritive and functional disturbances, which may be grouped according to their causes into septic and non-septic.

The term pericementitis has been indiscriminately applied to all affections of the pericementum, and in some cases erroneously, for in not all affections of this structure do the phenomena of inflammation appear. However, most of the acute and chronic degenerations are accompanied by evidences of inflammation.

Bödecker's division of the affections of the pericementum into purulent and non-purulent is misleading. Cases may be due to septic causes without pus-formation; pus-formation represents but one form of sepsis.

The most convenient clinical classification of these disorders is that offered by G. V. Black:[1] first, diseases of the pericementum beginning at the apex of the root; secondly, those beginning at the gum-margin; thirdly, those beginning in some intermediate portion of the pericementum. These may again be divided, according to their causes, into septic and non-septic. Another clinical classification would be into localized and general disturbances—another into acute and chronic.

Evidences of Pericemental Disturbance.—It was noted in the study of the diseases of the dental pulp that the diagnostic signs of pulp-disturbance were exaggerated or diminished response to thermal stimuli; reflected instead of localized pains; and, except in rare cases of advanced degeneration, no tenderness upon percussion. Disturbances of the pericementum are accompanied by entirely different symptoms which serve to distinguish between them and diseases of the pulp. They are, in general, tenderness upon percussion. As shown by Black,[2] the peri-

[1] *American System of Dentistry*, vol. i. [2] *Ibid.*

cementum is the touch-organ of the tooth, its tactile organ, through which a tooth locates force applied to the tooth. The pains of pericemental disturbance are, therefore, in the majority of cases, exactly localized, instead of not being localized as in the case of the pulp. A tooth tender upon percussion has its pericementum the seat of disturbance. Most cases of pericemental diseases are accompanied by vascular reactions ranging from an increased blood-flow or grades of hyperæmia, to pronounced inflammation, and have the corresponding symptoms. The increased volume of the pericementum causes the protrusion and loosening of the tooth, heightened sensitivity being the accompaniment. As the vascular supply of the pericementum and that of the gum are in a degree collateral (see Chapter VIII.), evidences of vascular engorgement are seen in the gum overlying the affected tooth. Owing to the altered density of the parts surrounding the tooth-root, percussion upon the tooth elicits a different sound from that observed in health—the sound is dull. The general symptoms of pericemental affections are, therefore, tenderness upon percussion and a dull percussion-note, more or less protrusion and looseness of the tooth, and a deepening of the local gum color.

Diseases of the Pericementum beginning at the Apex.

Diseases of the pericementum beginning at the apex of the root are of two classes, septic and non-septic. The septic cases are almost invariably the sequel to disease of the pulp, namely suppuration and gangrene ; or arise in consequence of infection through the canals of pulpless teeth. The non-septic cases are due to mechanical and chemical irritants, and in rare cases to undiscovered causes.

ACUTE SEPTIC APICAL PERICEMENTITIS—ACUTE ALVEOLO-DENTAL ABSCESS.

Definition.—By septic apical pericementitis is meant a condition due to the entrance and the multiplication of septic organisms in the apical pericementum. The condition may be acute or chronic, the chronic cases being usually a sequel to an acute septic pericementitis.

Causes.—By far the most common cause is infection of the pericementum, in the last stages of pulp-putrefaction, by pyogenic organisms. In the last stages of pulp-destruction through septic processes, it is usual to find that evidences of pericementitis exist : the tooth is tender upon percussion, is loosened, and protrudes slightly. As a rule, this irritation subsides after a few days. Succeeding this, is a period of quiet, before pronounced septic pericementitis arises. It appears as though the waste-products of the bacteria in the decomposing pulp acted as irritants upon the apical pericementum, and that by the formation of a

barrier of new tissue the pericementum was temporarily protected. Its causes are found in all of the conditions under which moist gangrene of the pulp occurs.

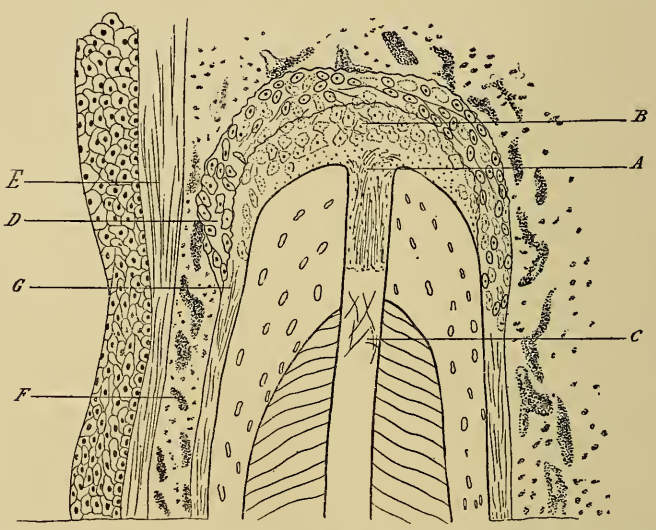
Purulent apical pericementitis is not always preceded by pulp-death and putrefaction; although when the condition arises the pulp, if alive, dies. Considerable purulent destruction of the pericementum may occur near the apex of the root and the pulp of the tooth remain alive. The path of infection in these cases is unknown; it is probably identical with that of pulp-putrefaction in cases without existing or previous caries. The possibility of infection by deposition of pyogenic organisms which have found their way into the circulation from other parts of the body must be admitted. The common infective organisms, those which predominate in purulent apical pericementitis, are the pyogenic staphylococci. Schreier[1] stated that out of twenty cases he had found in fifteen a diplococcus which he termed the diplococcus pneumoniæ. Miller's[2] experiments failed to confirm the identity of the diplococcus found with that of pneumonia. Schreier's studies[3] exhibit a preponderance of the staphylococcus pyogenes albus and aureus, diplococci, and occasional streptococci, virtually the same organisms that are found in the deeper portions of a suppurating pulp: this fact in itself is enough to show the continuity of infection from the pulp-canal. It is a well-known clinical fact that acute outbreaks of septic apical pericementitis are most liable to occur under those conditions when patients "take cold." Schreier points out that these atmospheric states produce a bodily condition which favors the development of the diplococcus pneumoniæ, and finds in the association of these factors the reason why this diplococcus should be pathogenic in the dental condition.

Morbid Anatomy and Pathology.—The general morbid anatomy of this condition is that of abscess, modified, of course, by the anatomical structure of the part. Pyogenic organisms gain access to the pericementum through the paths named, and a degree of inflammation is excited, governed by the virulence of the infection and the condition of the patient. An abundant, fibrinous, coagulable exudation is poured out into the interstices of the pericementum, not beneath the membrane—an exudation of leucocytes occurs; the pericementum swells, its fibres at and about the apex soften, the fixed cells of the tissue undergo proliferation, and the tooth is protruded and loosened. The inflammatory corpuscles are killed in great numbers by the waste-products of the organisms; the exudation is peptonized—liquefied by ferments excreted by the bacteria, the dead corpuscles being also broken down into a granular detritus. The inflammatory process extends radially from the focus of

[1] *Dental Cosmos*, 1893. [2] *Ibid.*, 1894. [3] *Ibid.*

infection, an inflammatory zone preceding the death and disorganization of corpuscles and effusions. The destruction of tissue proceeds in all directions, advancing most rapidly in the direction of least resistance, until the abscess reaches the surface, points, and discharges its contents. From the pericementum the inflammation extends to the alveolar bone, which is melted down molecularly; thence to the periosteum, which undergoes inflammatory degeneration; the gum-tissue is next involved,

FIG. 307.



Showing the morbid anatomy of septic apical pericementitis (acute): *A*, pus; *B*, area of dying leucocytes; *C*, foreign matter in root-canal; *D*, excavation of process (osteomyelitis); *E*, swollen periosteum and gum; *F*, alveolar bone; *G*, pericementum at edge of necrosis.

until it is softened and perforated. While in the vast majority of cases the direction taken by the pus and the point at which it finds exit is the buccal or labial aspect, and immediately over the root-apex of the affected tooth, or near it, these being the directions of least resistance, other anatomical conditions or histological peculiarities (see Chapter VIII.) may make the direction of least resistance in some other path.

Instead of the circumscribed suppuration described as the ordinary course of abscess-formation about the apices of roots (septic apical pericementitis) which accompanies infection by the staphylococci, clinical evidences of infection by a streptococcus occasionally appear. The inflammatory process, instead of being circumscribed, is diffuse; the inflammation extends along the lines of connective tissues and of lymphatics; the connective tissues are swollen, the swelling extending

to the tissues of the cheek, down the neck, and even to the shoulder—a phlegmonous inflammation. Instead of the comparatively free flow of pus which follows incision of the swelling in ordinary abscess, pus-formation in streptococcus infection is seen, upon incision, to be limited and sero-purulent. While in alveolar abscess of the ordinary types evidences of septic intoxication or poisoning are unusual, the lymphatics being blocked, as a rule, by the inflammatory exudation; septic intoxication and poisoning are the rule in the erysipelatous cases, those probably due to streptococcus infection; bacterial poisons being taken up by the lymphatics find their way into the circulation.

After spontaneous discharge of the pus from an abscess, the condition remaining is that of an ulcerous surface (the abscess boundaries), which is being continuously infected from the putrescent pulp-remnants. The conditions, it is seen, are not like those of ordinary abscess, where the infective material is largely discharged in the pus-evacuation, and the cells bounding the abscess-wall dispose of remaining bacteria, so that regeneration of tissue occurs. Spontaneous healing of an alveolar abscess is the exception; the embryonic tissue lining the abscess-walls being continuously infected, degenerates and dies instead of regenerating, leaving a condition known as chronic alveolar abscess, or chronic apical septic pericementitis purulenta.

Symptoms.—According to the severity of the symptoms, apical pericementitis may be divided into several grades, each of which represents more or less well-defined pathological conditions. It is to be remembered that the normal progress and outcome of this condition are toward the formation and evacuation of pus, so that the symptoms will be largely governed by the difficulty or readiness with which the discharge is effected. Infection represents the first stage of the acute disease, pus-discharge the last, after which the acute symptoms subside.

The first symptom to appear is tenderness upon percussion, the disease being ushered in by an active hyperæmia. As in other active hyperæmias, the sensitivity of sensory nerve-fibres is heightened; if the tooth be moderately pressed upon, it is tender; but if forcibly pressed upon—*i. e.*, the apical arteries be compressed—the hyperæmia is momentarily lessened, and the pressure brings a sense of relief. This period is succeeded by a protrusion of the tooth beyond its fellows; it appears to be, and is, longer than the other teeth; it is loosened and becomes very sore upon pressure, and soon tender or exquisitely painful to the slightest touch. Throbbing pain now occurs, and the gum overlying the affected tooth, first heightened in color, becomes swollen and deeply colored. These conditions correspond with the exudation-period of the inflammation. The tooth becomes progressively looser, and so tender

that it will not bear the slightest touch ; the throbbing pain increases in severity, and the gum-tissue, and, it may be, the tissue of the cheek or lip, also become much swollen. The swelling of the gum, at first of board-like hardness, softens at its highest point ; soon a yellow spot appears, the mucous membrane bursts, and a discharge of pus follows. As soon as softening of the swollen gum occurs the excruciating pain—an acute alveolar abscess is one of the most painful of diseases—and the tenderness of the affected tooth usually diminish, but some degree of protrusion and loosening remains.

In multirooted teeth the inflammation and abscess frequently appear on only one root. If the case be seen early, before the active exudation-period of the inflammation sets in, the symptoms may be clearly localized in one root, the tooth exhibiting tenderness upon pressure over the affected root, but not upon the opposite side.

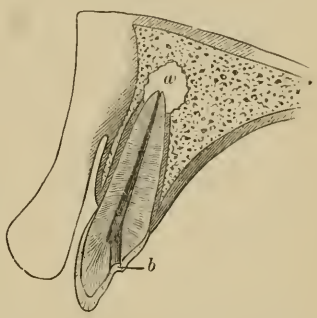
The symptoms above described are those of average severity. Variations occur ; some cases have a lesser degree of intensity, some a higher degree. Pain and swelling may be comparatively slight and pus-discharge prompt. In other cases pain, swelling, and looseness of the tooth are pronounced at an early period, and several grades of constitutional disturbance may appear. The pulse increases in volume and tension, the tongue is coated, and the temperature of the body rises ; the rise of temperature may be ushered in with a distinct chill—*i. e.*, a condition of fever is present, due to the absorption of bacterial products. As a rule, these general symptoms accompany the cases in which the vascular disturbance is widespread. Instead of the swelling extending but little beyond the overlying gum, the tissues of the lips, cheeks, or neck may be very much swollen and the eye of the affected side injected. In some cases the outer skin may become reddened and dusky, exhibiting the evidences of extension of the inflammatory process far from its original site.

Clinical History.—As shown in Chapter VIII., the apices of the roots of teeth lie nearer to the external alveolar wall than to the inner, with the exception of the palatal roots of the upper molars and the roots of the lower molars, the ends of the roots in some cases being covered by laminæ of bone of extreme thinness. Apparently the alveolar periosteum and gum-tissue vary in density. Recognizing these differences, the clinical history of acute alveolar abscess may be divided into three stages : first, that of initial inflammation and pus-formation ; secondly, the destruction of the alveolar process ; thirdly, the passage of pus through the periosteum and mucous membrane. The second stage is usually the longest. The duration of the disease depends upon the readiness with which the tissues between the point of beginning pus-formation and its exit yield. When the pulp-chamber is open pus may

find exit by this path, constituting the condition known as blind abscess —a misnomer, because a blind abscess is one without a point of discharge, without a fistula leading to it; in the cases discharging *via* the canal, the latter may be considered a fistula.

These cases usually run a short course, the inflammatory symptoms rarely being severe, and the tissue-destruction limited (Fig. 308). Notably upon lower molars, and upon the palatal roots of upper molars, the density and thickness of bone overlying the roots may make paths of greatly increased resistance, so that the destruction of tissue proceeds along the line of the pericementum, the pus finding exit at the neck of the tooth (Fig. 310). It is rare in cases of lower second molar, and still more rare upon the third molars, that pus finds exit over the apex of the root, the dense bone of the external oblique line (Fig. 309) offering the greatest resistance

Fig. 308.



Blind abscess at the root of an upper incisor: *a*, abscess-cavity in bone; *b*, drill-hole exposing the pulp-chamber for treatment. (Black.)

Fig. 309.

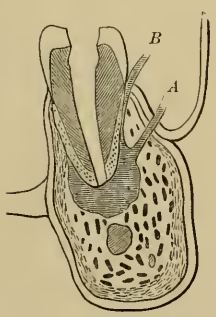


Fig. 310.

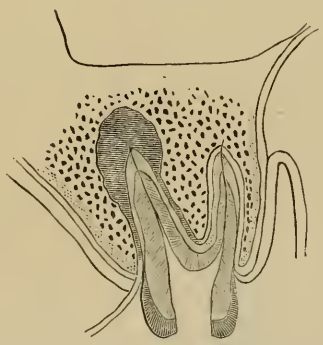
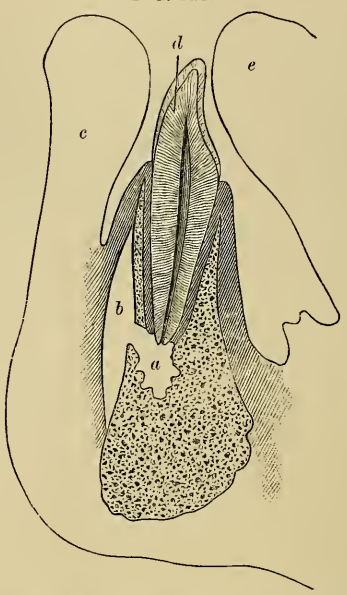


Fig. 309.—Abscess upon lower third molar, showing the usual paths of pus-exit, *A* and *B*.
Fig. 310.—Abscess upon palatal root of an upper molar discharging at the neck of the tooth.

Over any teeth the outer fibrous layers of the external periosteum may offer unusual resistance to the perforative advance of pus, so that when the fibres of attachment of the periosteum have been softened by the inflammation, and pus gains entrance between bone and periosteum, it may travel or burrow along the course of this membrane (Fig. 311), depriving the bone of its main nutritive source, so that limited necrosis threatens. The roots of the central incisors may lie unusually close to the floor of the nose, and be overlaid externally by an unusually resistant layer of bone; in these cases the path of least resistance may be in

the direction of the floor of the nose, the abscess opening at that point (Fig. 312).

Fig. 311.



Acute alveolar abscess of a lower incisor, with pus-cavity between the bone and the periosteum: *a*, pus-cavity in the bone; *b*, pus between the periosteum and bone; *c*, lip; *d*, tooth; *e*, tongue. (Black.)

The root-apices of the posterior upper teeth, particularly of the first and second molars, may after the age of twenty-five or thirty be

Fig. 312.

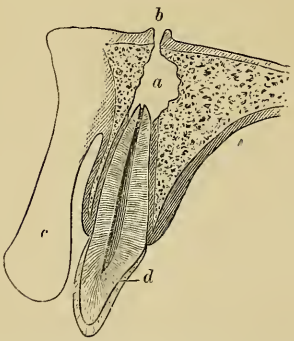


Fig. 313.

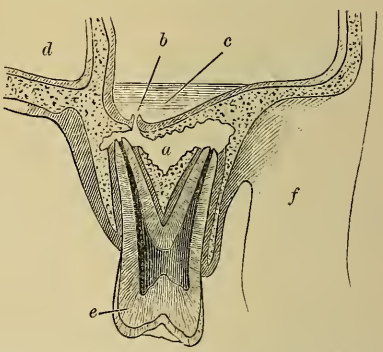


Fig. 312.—Alveolar abscess at the root of a superior incisor discharging into the nose: *a*, large abscess-cavity in the bone; *b*, mouth of fistula on the floor of nostril; *c*, lip; *d*, tooth. (Black.)

Fig. 313.—Alveolar abscess at the root of an upper molar discharging into the antrum of Highmore: *a*, abscess-cavity in the bone; *b*, mouth of fistula on the floor of the antrum; *c*, pus in the antral cavity. (Black.)

encroached upon by the enlarging maxillary sinus, so that any or all of the roots of these teeth may be separated from the floor of the sinus

by but a very thin lamina of bone; should abscess arise upon any of these roots, pus-discharge into the antrum would necessarily follow (Fig. 313).

Resort to the use of poultices, for the relief of the pain of abscess-formation, may induce such a softening of the tissues over which they are applied, that the passage of pus is invited toward the exterior; the abscess may thus open upon the face or neck, producing permanent, disfiguring scars.

In patients who are in a cachectic condition, who have an evil heredity, or whose tissue-resistance is markedly lessened in consequence of tuberculosis, or more frequently of syphilis, septic pericementitis may run a riotous course; the bone suffers extensively by direct action; the periosteum is undermined, is stripped from the bone over large areas, and breaks down readily; so that while in the healthy person alveolar abscess-formation may run a direct course and find prompt outlet, in the syphilitic patient extensive pus-infiltration, with necrosis, may occur. In cachectic persons lymphatic involvement is common; waste-products of bacterial origin find their way into the lymphatics and set up secondary irritative processes in the nearest lymphatic glands—lymphadenitis.

In persons whose oral hygiene is neglected the third stage of alveolar abscess is frequently violent and the inflammatory process widespread.

In acute abscess-formation the inflammatory action precedes the advance of pus, which furnishes a guide to the direction the pus is pursuing; viz., where the most intense coloration and the greatest swelling appear will be the point at which the abscess will point or discharge. A subsidence of inflammation without an immediately discoverable point of pus-exit should lead to the suspicion that the discharge has taken place in an unusual situation.

Diagnosis.—If a tooth have been the seat of acute septic pericementitis of high grade for twenty-four hours, pus has almost certainly formed, and its presence may be safely diagnosed. The symptoms rarely leave any doubt as to which tooth is affected, except where two contiguous teeth, evidently pulpless, are both loosened and surrounded by a zone of inflammation. Even in these cases there will be found differences in response to tapping or pressure which will indicate which tooth is the disease-focus.

Prognosis.—In the majority of cases the prognosis of acute alveolar abscess, as to the future retention of the tooth, is favorable; and usually very favorable, if the case receive intelligent therapeutic aid. The future of the tooth depends upon the thoroughness with which sources of infection may be destroyed and permanently removed, and the completeness with which regeneration of tissue can be induced.

If the first stage of abscess-formation be prolonged, destruction of pericementum is correspondingly increased; a prolonged second stage causes an increased molecular destruction of alveolar bone; a prolonged third stage may mean stripping of the periosteum from the alveolar wall, or infiltration of pus into the connective tissue of the lip or cheek. Marked swelling of the tissues of the face, with an increasing redness of surface, leads to the suspicion of pus-presence and the danger of its external discharge. Rigors and fever, appearing during the course of the inflammation, are evidence of absorption of and the presence in the circulating fluids of bacterial products. Repeated rigors, with pronounced depression, diarrhœa, and delirium, indicate that pyæmia exists. Delay in the natural evacuation of the pus should lead to the suspicion that it is pursuing an unusual course, its direction being usually determined by the focus of inflammatory action.

Treatment.—The first principle of treatment is the removal of the source of infection. As in all other septic diseases, there is no means comparable with this in point of effectiveness. After removing the source of infection the symptoms of the disease-process subside rapidly. If it be not removed surgically, the disease persists until the pus finds vent, when the inflammation subsides. The immediate accomplishment of this end may, however, be impracticable in some cases. Recognizing the putrescent pulp-canal contents as the source of infection, primary attention is, of course, directed toward sterilization and removal of these contents. If the case be seen before the inflammatory process become pronounced, entrance to and cleansing of the canals can usually be accomplished. If the pulp-cavity be open in such cases, direct approach is made to the canals through the carious cavity. A free syringing with strong solutions of meditrina precedes the opening of the canals. If it be a filled tooth, and the filling is in a situation that by an opening made through it, or by its removal, direct access to the canals can be gained, the opening should be made. The opening of the cavity is to be accomplished by means of a very sharp and small spear-point drill revolving in a perfectly true hand-piece. Large drills, ill-sharpened and in worn hand-pieces, produce a jarring which adds notably to the tenderness of the pericementum. According to the amount of tenderness, the tooth will require a counter-pressure to that of the drill. If the entrance be made through the occlusal face of the tooth, or in a direction which would cause direct upward pressure on the apical pericementum, a ligature of linen thread with long ends may be placed around the tooth,[1] and traction be made by drawing on the loose ends of the ligature. Effective counter-pressure against lateral entrance to the pulp-chamber may be made by softening a small roll of modelling compound and

[1] J. Foster Flagg, *Lectures on Dental Therapeutics.*

moulding over the face of the affected tooth and several of those adjoining it, and permitting it to harden. This temporary splint is held in place by the index finger of the left hand. In case the inflammatory process is marked, it is frequently necessary to make a vent-opening by the most direct path—*i. e.*, at the junction of enamel and cementum—directly into the chamber.

As soon as entrance to the pulp-chamber is effected, the cavity is syringed with a strong antiseptic; a 20 per cent. solution of meditrina answers well in this connection. Fine probes are passed and repassed into the opening to free the outlet, so that gases may escape and fresh portions of the antiseptic be worked into the cavity. The quickness with which relief is secured will depend upon the thoroughness with which the canals are entered and their putrid contents destroyed. A tedious class of cases are those in which a canal of a molar is unfilled or but partially filled. Unless entrance to and cleansing of the canal be accomplished, the inflammation will proceed until the pus finds external vent. An hour spent in gaining access to and cleansing such canals is well spent.

If entrance to the canals is free, repeated applications of sodium dioxid solutions should be made, pumped into the canals, and the cavity washed from time to time with meditrina or hydrogen dioxid. Near the end of the canal the meditrina is used alone with broaches, and finally by syringing. The canals are dried, and an anodyne antiseptic, such as a mixture of thymol and menthol dissolved in glycerin, is pumped into the canals. If now provision for surgical rest of the irritated pericementum be made, relief is tolerably certain. A moldine impression is taken of the adjoining tooth, if a bicuspid or a molar, or of one the bicuspids, if a labial tooth be the one affected, and a fusible metal die is made. Driven into a block of soft lead, a counter-die is formed and a metal cap to cover the occlusal and part of the buccal and lingual surface of the tooth may be swaged in a few moments. About No. 26, American gauge, should be the thickness of the metal. The tooth is dried and the cap attached by means of zinc phosphate, and allowed to remain for a day or two. This will insure rest of the affected pericementum. If now the gum, at a distance from the tooth, be painted with tr. iodin and chloroform as a counter-irritant, the inflammation usually subsides and almost disappears in a couple of days. These several measures are to be regarded as the abortive treatment of alveolar abscess; they apply to all cases if seen early enough, and will in the majority of cases prevent the disease of the pericementum passing the early inflammatory stages. In all cases the severity of the inflammatory process is lessened in proportion to the thoroughness with which the antiseptic measures are applied.

If the case be a more severe one, or at a later stage than that described, the excessive tenderness of the tooth may preclude any attempt at drilling into the pulp-chamber without the administration of a general anæsthetic. When the cause, clinical history, and indicated therapeutics in alveolar abscess, are viewed, there can be no two opinions as to the wisdom of anæsthetizing the patient and effecting an entrance to the pulp-canals. It cannot be too strongly emphasized that canal-sterilization is in order at any stage of abscess-formation, as is also the free and frequent use of antiseptic mouth-washes—pyrozone and meditrina. Failing to administer a general anæsthetic, the canals are opened as freely as the tenderness of the tooth permits; the use of any instruments, except *broaches* applied with the utmost delicacy of touch, is precluded by the intolerable pain. An effort is made to limit the extent of inflammatory action. After the early stages and up to nearly the point of pus-perforation, hot applications in the mouth and the use of counter-irritants to the gum but provoke the inflammatory condition. The most effective measure is local bloodletting by means of a leech, if possible, or by making several free cuts with a bistoury, which tend to relieve the engorged vessels of the pericementum. The mouth should be washed with warm antiseptics before and after the incisions. Dry cups to the back of the neck and hot mustard foot-baths are also useful derivative measures.

In the still more severe cases marked relief of the inflammatory symptoms and the pain follows the administration of 10 gr. of Dover's powder, in addition to the measures advised. A saline cathartic, magnesium sulfate, should be administered the next morning, with a view to relieving the constipation following the use of the opium, and to act as a derivative by inducing free watery stools.

If high inflammation persist for more than twenty-four hours, pus is almost certainly present in the pericementum, and possibly more or less molecular destruction of bone has occurred. In rare cases, where the bone-covering of the root-apex is extremely thin, the pus may be at this time at the point of exit. The dictum of surgery, to give vent to pus as soon as it is discovered, applies as well in the condition under discussion as anywhere else. When it is considered that prior to evacuation of the abscess destruction of tissue is going on in all directions, it is evident that tissue-destruction, complications, and the possibility of septic intoxication, or even more serious general disturbances, will be averted by gaining quick access to the focus of infection and removing it. Some operators advise that an artificial opening be made even in the earlier stages of acute septic pericementitis, recognizing that the case is septic and that radical relief is only secured through complete antisepsis. If the case be seen early, however, the abortive measures previously de-

scribed can be instituted. The old practice of waiting until the pus has penetrated the alveolar periosteum is unsurgical.

To insure quiet of the patient while opening into the apical space from the gum, it is advisable to administer nitrous oxid, if this agent be available. The mouth should be freely washed with strong antiseptics and a cut made through the gum over the apex of the affected root. The incision is permitted to bleed, and is then packed with cotton containing phénol sodique. As soon as bleeding has ceased the nitrous oxid is administered; if it be not at hand, a pellet of cotton containing a 10 per cent. solution of cocain is laid against the periosteum. The first cut, made with a stout-pointed bistoury, penetrates to the bone. A sharp scaler or chisel is used to scrape away a small area of the alveolar periosteum; next a spear-pointed drill, revolving rapidly, is quickly passed through the outer alveolar plate into the apical space. The apices of the roots lie, as a rule, slightly higher than the line of mucous membrane reflection. The cut should be free and the periosteum scraped away, to avoid annoying and disfiguring emphysema of the cheek-tissues, which may occur if these precautions be not taken. The pain following the operation may be relieved, after bleeding has ceased and the cavity has been washed out with warm antiseptics, by pushing a crystal of cocain hydrochlorid into the cavity as far as it will go. As soon as the inflammatory symptoms have subsided sufficiently to permit working upon the tooth, the pulp-canals are to be opened and sterilized.

The case may not be seen until its third stage, when the pus is in the tissues exterior to the alveolar process. In these cases a very sharp bistoury is passed into the swelling at its most prominent part and a deep and free incision made. In case the inflammation have extended to the tissues of the cheek, an outcome most to be feared in abscess upon the lower third molar or upon the upper first or second molar, antiphlogistics should be applied to the cheek—

℞.	Plumbi acetat.,	ʒj;
	Tr. opii,	ʒj;
	Aquæ,	Oj.—M.

Compresses wet with this preparation are laid upon the cheek, and a free, deep incision made in the gum at the junction with the cheek. An examination should always be made of the palatal and lingual alveolar aspects, to note whether the inflammatory and suppurative process tends to take either of those directions.

If, in connection with the lower third molar, marked swelling is observed in the submaxillary triangle, free incision of the tissues of the

floor of the mouth should be made at the angle of junction with the bone. The cut should be made close to the bone and into it, but not too deep, lest the mylohyoid artery or nerve be injured. In all cases which threaten to open externally the antiphlogistic compresses are to be continuously applied externally, and after incision stimulant mouth-washes should be used; that recommended by Prof. Garretson is excellent—tr. myrrhæ et capsici in water.

It is ever to be borne in mind that so long as the source of infection remains pus-formation continues, and so long as pus forms, tissue-destruction is in progress; furthermore, in proportion to the amount of tissue-loss perfect recovery after alveolar abscess is delayed or imperfect.

While it is the clinical experience of nearly every operator that a tooth and adjacent structures may recover from inflammation which involves not only the first tooth attacked, but by an extension of the inflammatory process involves the general periosteum and neighboring teeth, provided the case receive prompt and decisive surgical treatment, yet the danger of septicæmia in prolonged cases is always imminent. When the general periosteum is involved, as shown by extensive boggy swelling in the mouth, if several free incisions carried to the bone do not afford prompt relief, the tooth which is the centre of infection should be promptly extracted. If, in the course of the pericementitis, chills, followed by fever, a coated tongue, and much physical depression occur, a general infection is to be feared, and no time should be lost in sterilizing the mouth, extracting the tooth, and subjecting the socket to free spraying with antiseptics.

CHAPTER XXII.

SEPTIC APICAL PERICEMENTITIS (CHRONIC).

Chronic septic apical pericementitis exhibits several grades as to its extent and effects; one of the more pronounced types or grades being the direct outcome of acute apical pericementitis, resulting in pus-formation, a condition known as chronic alveolar abscess. After the discharge of pus, either spontaneously or through surgical aid, as noted in the discussion of acute alveolo-dental abscess, the source of infection remains, and pus-formation continues as a chronic process, which rarely disappears spontaneously; the acute inflammatory symptoms, however, subside and do not reappear unless there is some interference with the escape of the pus.

Clinically chronic abscess presents itself in two classes: cases without a fistula communicating with the mouth-cavity or other part, and those in which the pus finds vent through a fistula.

Chronic Alveolo-dental Abscess without Fistula.

Pathology and Morbid Anatomy.—Although the acute inflammatory symptoms may subside and be replaced by those of atonic hyperæmia, as soon as pus finds vent through the canal of the tooth; pus-formation—*i. e.*, tissue-destruction—proceeds radially from the affected root. The conditions existing immediately after evacuation of the pus are a denuded root-apex, about which are peptonized (liquefied) effusions, in which are shreds of dead tissue, and dead, dying, and disintegrated inflammatory corpuscles, occupying a cavity bounded by embryonic tissue, which is being gradually invaded by pyogenic organisms; the surrounding tissue is being transformed into or replaced by embryonic tissue, the result of inflammatory degeneration. The cavity bounded by this wall of embryonic tissue is constantly increasing in size. In long-continued cases organization of the boundary-wall may occur, and the cavity be enclosed by a capsule of vascular fibrous connective tissue. If teeth be extracted at this stage, this fibrous sac may come away with the tooth; it is the structure sometimes termed a pyogenic membrane.

The influence of gravity has much to do with the direction of

tissue-destruction in chronic abscess (Figs. 314 and 315). In the lower jaw the tendency is to burrow into the cancellated tissue of the bone

Fig. 315.

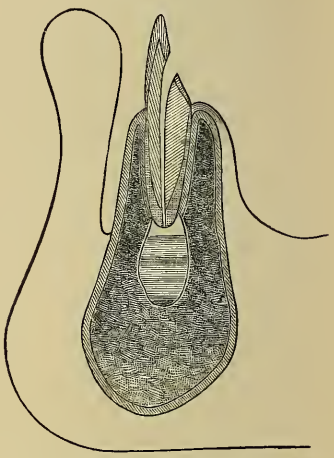


Fig. 314.

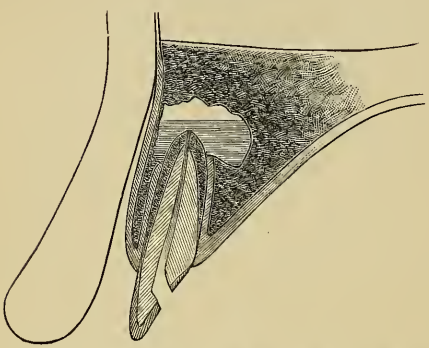
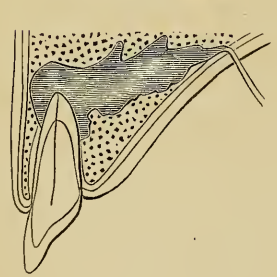


Fig. 314.—Chronic blind abscess of upper incisor, showing tendency of pus to progressively destroy pericementum owing to the influence of gravity.

Fig. 315.—Chronic blind abscess upon lower tooth, showing tendency of pus to sink into the substance of the lower maxilla, owing to the influence of gravity.

away from the tooth, so that destruction of the pericementum may not be very extensive. In the upper jaw the tendency is to spread along the pericementum and into the cancellated bone, so that the cavities of chronic abscess upon the upper anterior teeth particularly may cause extensive excavation in the palatal process of the superior maxillary bone (Fig. 316). The pus may burrow in irregular and circuitous directions, until it finds external vent. In long-established cases the denuded root-apex becomes the seat of calcic deposits.

Fig. 316.



Symptoms and Diagnosis.—Attention is directed to some pulpless tooth or to a crownless root, around which the gum-color is deepened and which is more or less loose, indicating softening of its pericementum, but no fistula is present. After isolation under rubber-dam pus may or may not be discoverable in the canals. If pus is seen, the diagnosis is evident. If pus be not seen, and the canals be drenched with a 50 per cent. solution of sodium dioxid, the application being continued until it is reasonably certain that all infective material in the canals has been destroyed, the canals may be closed with the dressing usual in such cases, twists of cotton saturated with an antiseptic oil, when irritation should be allayed if an

abscess does not exist; but if, after some hours, or a day or two, inflammatory symptoms arise, the presence of pus should be suspected, and its existence may be confirmed by its flow upon removing the canal-dressing. More or less pus should always be suspected about the roots of teeth which are crownless or pulpless, and have unfilled canals, when the tooth is loose, and the overlying gum injected. In doubtful cases canal-fillings, even after thorough cleansing with sodium dioxid, are made of an easily removable material, and are only made tentatively until it is certain that a tightening of the pericementum and a fading of the deepened gum-color, instead of an increased irritation, follow their insertion.

Prognosis.—The fate of the affected root depends upon the amount of tissue-destruction, the length of time suppuration has been going on, and the recuperative powers of the patient's tissues, together with—most important of all—the thoroughness with which infection and the sources of infection are removed. If the last-named object can be attained, astonishing recoveries occur; in its absence, the tooth is certain to be permanently crippled and to be a menace to the surrounding tissues. It is remarkable, however, how long pus-formation may continue about the root of a tooth, and cause comparatively little disturbance, except in a very circumscribed field. The presence of pus in any cavity of the body should never be regarded as without danger; and, doubtless, constitutional effects from pus-formation about the teeth are often present without the operator being cognizant of them.

Treatment.—The principle of treatment is to remove all pus, dead tissue, and infective organisms, induce a regeneration of tissue to obliterate the abscess-cavity, and prevent future infection. The heroic and most successful method of treatment is to remove, as a primary measure, the mechanical impediment to the thorough washing and sterilizing of the abscess-cavity. So long as the entrance to the abscess-cavity and the exit from it are but the constricted passage of a root-canal, it is evident that the complete filling and emptying of the abscess-cavity will be attended with difficulty, depending upon the size of the canal and of the abscess-cavity. If, however, an external fistula exist, the complete washing of the tract and emptying of the cavity are much facilitated; abscess with external fistula is much more amenable to treatment than when no such exit exists. The tendency of modern practice, therefore, is to establish a free artificial fistula in all cases of so-called blind abscess.

The canals are cleansed with sodium dioxid solution, and the canal length and direction recorded upon a broach. This length is measured upon the gum to determine the point of entrance. The most certain

and quickest method of making the proper entrance is to administer nitrous oxid, or, if that be not available, to inject a few drops of a boiled solution (10 per cent.) of eucain into the gum and remove a cylindrical portion of gum-tissue by means of a Rollin's tubular knife, or make an incision to the bone and scrape off a portion of periosteum; entrance to the abscess-cavity may then be made by means of a drill or small trephine (Fig. 318). The case is now treated as one of abscess with fistula (which see).

Fig. 317.

4 6 5

Tubular knives.

Fig. 318.

1 2

Walker-Younger trephines.

Cases may be treated without making an artificial fistula, but the results are rarely so satisfactory and the cure seldom so complete as when an artificial opening is made. The best results are obtained where the destruction of tissue has been very limited, where the pus has found exit through the canal at an early period, and where the case is seen soon after subsidence of the active inflammation.

The tooth-cavity is cleansed mechanically, syringing freely with meditrina or 3 per cent. pyrozone solution, and the rubber-dam adjusted. The canals are cleansed by repeated applications of sodium dioxid solution, continued until the apex of the root is passed, when the canals are forcibly syringed with an acid solution of hydrogen dioxid until effervescence ceases. The canal is now thoroughly dried and filled with an antiseptic; Black's 1–2–3 mixture, campho-phénique, oil of cassia, and thymol are all useful. If the canals have been well cleansed and the antiseptics employed have been carried into the abscess-cavity, further use of antiseptics is not absolutely necessary, but it is prudent to use them to complete a possibly partial sterilization. The antiseptic may be driven in spray into all portions of the abscess-cavity, by blowing a blast of air into the canal through a chip-blower. The canals are next partially dried and loosely filled with cotton, and the crown-cavity hermetically sealed.

If in the course of two or three days, the tooth appears tighter and the vascular symptoms in the gum subside, a cure may be anticipated. As a precautionary measure, the tooth may be placed under rubber-dam, the seal and canal-filling removed, and the condition as regards odor and the presence of pus or blood-stains noted. If odor be present, a second cleansing with sodium dioxid should be practised as before, the canals

dried, and a cotton twist saturated with thymol packed into them. If no symptoms arise, this dressing may remain a week or longer. At the expiration of this time the abscess-cavity is probably filled with organizing granulation-tissue. The canal may be opened under extraordinary antiseptic precautions and filled with melted paraffin and aristol, nosophen, euthymol, or iodoform. The crown-cavity may be filled with zinc phosphate, and the case be dismissed for six months or longer. If a gold filling is indicated, this time should elapse before it is inserted.

If the tooth rebels against closing after more than one cleansing with sodium dioxid, it is scarcely worth while temporizing—an artificial fistula should be established.

In rare instances a cure of even extensive suppuration, with large cavity, may be effected without an artificial fistula, by sterilizing the affected root, aspirating the pus, washing out the abscess-cavity, and inducing regeneration. The tooth should be isolated, its canals cleansed with sodium dioxid and washed. As deep into the canal as possible the nozzle of a large syringe is placed; temporary stopping is packed into the crown-cavity tightly about the syringe; the piston is withdrawn, drawing the pus into the syringe. The withdrawal may fail entirely owing to the apical foramen being blocked by shreds of dead tissue. If the attempt be successful, the stopping is removed, the canals dried, and a drop or two of a 25 per cent. solution of pyrozone pumped into the abscess-cavity, more being added until effervescence ceases. When the canals are dried they are filled with an antiseptic—thymol, camphophénique, etc.—driven in spray into the abscess-cavity by blasts of warm air. If the evidences of disturbance subside, the case is treated as in the former instance. If the tooth rebel against closing, an artificial fistula should be established.

Chronic Alveolo-dental Abscess with Fistula.

Morbid Anatomy and Pathology.—The conditions found attendant upon this disease depend, first, upon the length of time elapsing between the inception of the inflammatory process and the evacuation of the abscess; secondly, upon the surgical anatomy of the parts; and, thirdly, upon the physical condition of the patient. In the majority of cases the pus from acute abscess finds exit almost immediately over the apex of the affected root, in the course of a few days, so that at this time a canal lined with embryonic connective tissue may connect with a cavity of any size lined with embryonic tissue, the walls of which continue in a state of infection. If the process continue, organization of the abscess-wall occurs as in abscess without fistula.

Instead of finding exit by a direct path to the exterior, the pus may burrow along the length of the pericementum and discharge at the neck

of the tooth. One-half or more of the lateral aspect of the pericementum may remain vital, although involved in a chronic inflammation, the remainder being destroyed. Where the apices of the roots of upper posterior teeth lie in very close proximity to the floor of the antrum, perforation of this floor may occur before tissue-destruction has proceeded far enough in other directions to afford escape to the pus. Extensive pus-accumulations may occur in the antrum in consequence. Other paths of pus-exit are noted in Chapter XXI., in connection with acute abscess; at such points the discharge may remain persistent. In some cases, after the subsidence of the acute inflammatory symptoms, pus-discharge may lessen and finally cease, the fistula healing, although pus-formation in the substance of the bone continues. The discharge only ceases, however, when the pus finds some other point of discharge; usually this is through the canal of the affected tooth; the condition then becomes one of blind abscess. The burrowing of the pus continues, however, and at a late period may find exit at a distant point. Upon a lower tooth, particularly the incisors, the pus may burrow downward through the cancellated tissue of the bone and emerge at the base of the bone and open upon the face (Figs. 319 and 320).

In other cases the pus may perforate the bone, and find passage along the submuscular tissue of the depressor muscles of the lip, opening above or under the point of the chin. The apices of the roots of teeth lying beneath the line of insertion of the mylohyoid muscle may cause an abscess to open in the neck-cavity. Cryer records a case where an abscess opening upon the face immediately anterior to the line of the facial artery, was traced to the root of a lower molar;

FIG. 319.

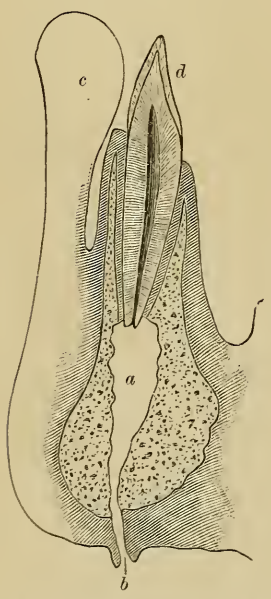


FIG. 320.

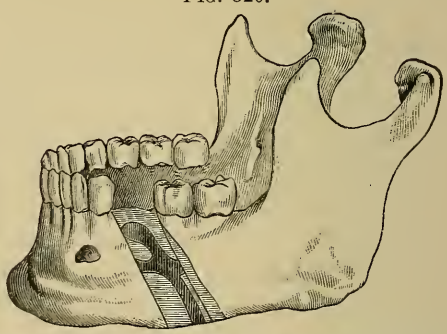


Fig. 319.—Chronic alveolar abscess of the root of a lower incisor, with abscess-cavity passing through the body of the bone and discharging on the skin beneath the chin: *a*, very large abscess-cavity; *b*, mouth of the fistula. (Black.)

Fig. 320.—Fistula passing down through the body of the lower maxilla. (Black.)

the direction of the sinus is shown in Fig. 322. In a case having a similar anatomical association, the pus penetrated the bone lingually, was encapsuled beneath the internal pterygoid muscle, and appeared as a swelling at the inner aspect of the angle of the jaw. Occasionally the apices of the roots of lower molars are separated from the inferior dental canal by only a thin lamina of bone, so that discharge into this

Fig. 321.

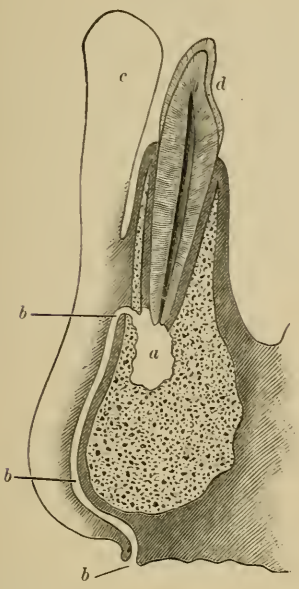


Fig. 322.

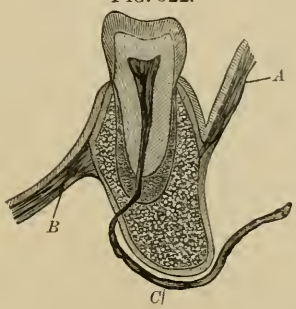


Fig. 321.—Chronic alveolar abscess at the root of a lower incisor, with a fistula discharging on the face under the chin: *a*, abscess-cavity in the bone; *b*, *b*, *b*, fistula following in the periosteum down to the lower margin of the body of the bone and discharging on the skin. (Black.)

Fig. 322.—Abscess with tortuous sinus, opening upon the face: *A*, tissue of cheek; *B*, floor of mouth; *C*, abscess-tract.

canal may occur with infiltration along the vessels and nerves in the canal. While discharge into the nasal chamber is most frequently associated with abscess upon the central incisors, abscess upon molars may discharge into the same cavity.

Symptoms and Diagnosis.—The symptoms of the condition are a fistulous opening in the gum or some other part, in proximity to or connected with a pulpless tooth. The tooth may have an open cavity, contain a filling, bear an artificial crown, or be free from caries. In other cases, the root which is the centre of infection may lie buried in the gum and be invisible. The source of the trouble in rare cases may be an impacted tooth (see Chapter X.). As a rule, the seat of the affection is indicated by sluggish vascular disturbance in the gum overlying the offending root. Any fistula existing in the maxillary region, either within or without the mouth, should be suspected to have originated in a septic pericementitis of some tooth. A soft silver probe should be passed along the tract to determine its direction, and, if possible, which tooth is affected. As a rule, such a tooth exhibits objective evidences of abscess, and the patient will give a history of subjective symptoms —those of inflammation of pericementum. If the tooth indicated

as the affected one be free from caries, the thermal test should be applied to determine the degree of vitality of the pulp. Should the tooth not respond to applications of cold, it is possible it may offer slight response to applications of heat. It is next examined by light reflected from the ordinary, or, better, the electric mouth-mirror, when, if the pulp be dead, opacity of the crown will be detected. An abscess upon an upper incisor opening upon the nasal floor may cause a discharge simulating that of ozæna. An examination of the nose will reveal a teat-like elevation upon the mucous membrane covering the nasal floor and an incisor beneath will be found carious and having a putrescent pulp, or, if non-carious, there will be a history of traumatic pericementitis and upon inspection an opacity. It may be mentioned here, in connection with death of the pulp from traumatism, that continuous thread-biting, and biting very hard substances, such as pieces of ice, nuts, etc., may cause death of the organ, presumably by thrombosis.

It is possible that the direction taken by a probe passed into the fistula will point away from the teeth present, passing into a space from which a tooth has been extracted. In that event the presence of a root-fragment, or piece of necrosed process, may be suspected.[1] If the neighboring teeth be excluded as causes of the inflammation, there should be no hesitation in making an exploratory incision down to the end of the probe which has been passed into the fistula; cases of dentigerous cysts have been detected in this manner. This condition, however, should be suspected when there is an absence of a tooth or teeth from the arch, no evidence, past or present, of pericementitis in any of the teeth of the arch, and a cystic tumor present in the jaw, or a fistula discharging upon the face after a history of maxillary periostitis. Caries or necrosis, although in many cases the result of septic apical pericementitis, may exhibit fistulæ opening into the mouth, without evident connection with the teeth. As a rule, cases of necrosis exhibit marked evidences of chronic inflammation of the tissues overlying the dead or dying bone; there are usually several fistulæ discharging from it. Caries may present but a single fistula and closely simulate ordinary alveolar abscess. Diagnosis is made by passing an excavator through the fistula. Dead bone is readily detected by touch; it has a rotten feel. In caries the instrument may be passed through the dead bone in various directions, and a characteristic sound be elicited by tapping upon it. Careful examination of the teeth must be made in all of these cases to determine the condition of the pulps and pulp-canals. In passing an instrument through a fistula to the apex of a tooth-root, where the disease-process has been of long duration, it may be found

[1] See case of Black's, *American System of Dentistry*, vol. i.

that the apex of the root is denuded of pericementum and roughened; that is, the apical cementum is necrotic; foreign deposits may be detected occupying portions of the necrotic area.

Treatment.—The first step of treatment is the thorough eradication of the source of infection. The tooth, filled or unfilled, bearing artificial crown, or no matter what its condition, should have its root-canals freely opened to their apices. The presence of a canal-filling should not hinder the complete opening, as the canal may appear to be filled, and in reality be imperfectly filled. If the abscess be upon a root which cannot be utilized by crowning, the root should be at once extracted. Impacted teeth should be removed by surgical operation. If the centre of suppuration be dead bone, it should be removed by like means.

In cases where there are useful tooth-roots, the tooth should be isolated under rubber-dam, and saturated sodium dioxid solutions be pumped into the canals until it is certain they are cleansed and sterilized. Fine canal-cleansers are then passed through the apex of the affected root, opening into the abscess-cavity. The canal and cavity are now freely syringed with 3 per cent. pyrozone until the contents of the abscess-cavity are driven bubbling through the fistula. This forcible irrigation is continued until the fluid comes away clear and without bubbling. M. L. Rhein[1] advises that a solution of mercuric chlorid in hydrogen dioxid be used for this washing. The wall of embryonic tissue lining the abscess-cavity being frequently infected with organisms which the hydrogen dioxid alone fails to kill, mercuric chlorid is added to destroy them. The same writer[2] advises that a positive electrode, a fine wire of chemically pure zinc, be inserted to the bottom of the canal and the current from a cataphoresis apparatus applied. By electrolysis, zinc oxychlorid is formed at the positive pole by the action of chlorin and oxygen, liberated from the fluids of the part, acting upon the zinc; the zinc oxychlorid in its nascent state is forced cataphorically into the walls of the abscess, where it acts as an effective antiseptic and induces the formation of granulation-tissue; hence the regeneration of tissue.

The usual practice is to fill the root-canal with some powerful antiseptic, such as campho-phénique, the 1–2–3 mixture of Black, or one of the antiseptic oils, and, by pumping with a smooth broach, force the antiseptic through the abscess-cavity and fistula. Fluids may be induced to flow an astonishing distance if the pumping be persisted in. They will make their way along such tracts as shown in Figs. 319, 321, and 322.

Their flow may be induced in some cases by dipping a sterilized

[1] *Items of Interest*, 1897. [2] *Ibid.*

rubber cleaning-cap (Fig. 323) in mercuric-chlorid solution and pressing it firmly upon the gum over the fistula, then relaxing the pressure until the cap rises and draws the antiseptic in the canal, along the abscess-tract.

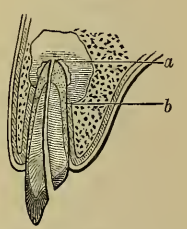
Fig. 323.

The canal is dried and filled with the paraffin-aristol mixture, or with thread dipped in an antiseptic oil, and the crown-cavity hermetically sealed. Nature should now complete the cure by obliterating the abscess-cavity with embryonic tissue, which subsequently organizes. In twenty-four hours but a slight serous flow should be noted, the patient having been directed to use antiseptic mouth-washes freely. In about three days no exudation should be present, and in a week the fistula should be healed. Unless a pus-discharge be observed, the tooth should remain sealed for a week, or, better, two weeks, and if filled with paraffin may remain permanently sealed. If pus-discharge appear, the tooth should be opened and treated as before.

If the case be one where an artificial fistula has been established, the wound should be kept from healing, after the antiseptic washing, by packing it daily with nosophen-gauze.

If a serous discharge persist longer than a week, a root-apex denuded of pericementum, saturated with noxious material, and probably encrusted with deposits of calculi, may be suspected (Fig. 324). A sterilized excavator should then be passed into the fistula and over the apex of the root, to discover the amount of denudation and the presence of deposits. If deposits be discovered, the root should be solidly filled; and nosophen-gauze be forced into the abscess-cavity, distending the fistula; if the latter be small, it may be enlarged by an incision and then packed. As soon as free exposure of the root is obtained a fissure-bur may be used to cut off the portion of root projecting into the abscess-cavity; the cut edges of the root may be rounded by means of scalers. It is a serviceable measure to scrape the abscess-walls in these cases, removing the debilitated and degenerating tissue and inducing healthy granulation. The cavity is packed with cotton and phénol sodique until bleeding ceases, and a packing of nosophen-gauze is inserted, to be renewed in daily lessening amount until the abscess-cavity is filled with granulations. The patient should use freely antiseptic mouth-washes. Each fresh packing should be preceded by an antiseptic douche.

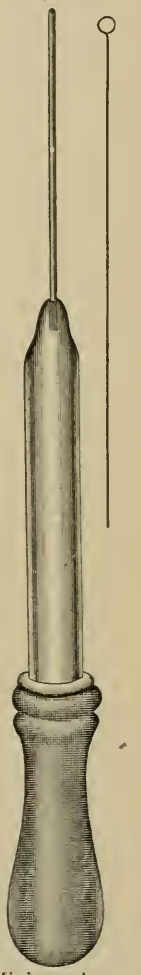
Fig. 324.



Chronic abscess, showing denudation of apex of root (*a* to *b*), with deposits of calculi (*a*) upon cementum.

In some cases of anomalous root-form, such as a sharp bend upon the upper end of the root, which renders it impossible to gain access to the apex of the root even with the aid of sulfuric acid, it may be necessary to treat the abscess through the fistulous opening. The roots should be sterilized and cleansed to as great a depth as possible with the aid of sulfuric acid and fine cleansers, and an endeavor made to force hydrogen dioxid through the apical foramen and out of the fistula by means of a syringe. The cavity of the crown should be filled with pink gutta-percha, and through it the nozzle of a syringe filled with 3 per cent. pyrozone thrust well up the canal. When the piston of the syringe is forced down, the solution may appear at the opening of the fistula, or it may fail to penetrate the foramen, and the backward pressure may force the gutta-percha from position. In that event myrtol should be placed in the canal, which should be filled with thread holding the same material. Three per cent. pyrozone should then be injected into the abscess-cavity through the fistula until effervescence ceases. The nozzle of a minim syringe (Fig. 325), charged with campho-phénique, or the 1–2–3 mixture, is then passed into the abscess-sac, and a couple of drops deposited. In very many cases the abscess will then proceed to recovery. The treatment should be repeated, if necessary. If several dressings applied at intervals of a week do not cause a disappearance of pus-formation, amputation of the offending portion of the root will be necessary. A heroic method of treating chronic abscesses which obstinately refuse to heal is by extraction and replantation. The method applies only to single-rooted teeth, although it has been successfully performed upon molars. The patient's mouth is to be sterilized, and the tooth extracted. The tooth is immediately placed in a solution of 1 : 1000 mercuric chlorid at a temperature of 120° F. It has been repeatedly asserted, but without satisfactory demonstration, that the cells of the deeper layer of the pericementum and the cementoblasts, and also the cement-corpuscles, retain their vitality for some period after extraction, and immediate replantation results in a re-establishment of the physiological union between the teeth and alveolus. It is certain, however, that measures which are necessary to thoroughly sterilize the tooth before its reinsertion would be fatal to any cellular vitality

Fig. 325.



Minim syringe.

which might exist in the cementum and its covering. The pulp-canal is opened from its apex and cleaned out with canal-cleansers, and a 25 per cent. solution of pyrozone placed in the canal, where it is allowed to remain for some time. In the meantime the socket from which the tooth has been removed is syringed with pyrozone, and should the pericementum not adhere to the tooth the depth of the socket is scraped by means of large spoon-excavators to remove the tissues implicated in the abscess. The cavity is washed with pyrozone, and a pledget of cotton, which has been dipped in campho-phénique, is placed in the socket at its bottom. The tooth is dried by means of warm air; the soft tissues at the apex, if any be present, are cut away for about one-eighth of an inch. The canal is filled with gutta-percha or filled solidly with gold, the end of the root cut off as far as it has been denuded of pericementum, smoothed, and returned to the antiseptic solution. The cotton is removed from the tooth-socket, which is syringed with 3 per cent. pyrozone, and the tooth returned to position. It is tied to the adjoining teeth by means of silk ligatures or held in place by an appropriate retaining appliance.

A chronic abscess may discharge into the maxillary sinus for a long period before being discovered, unless the pus-accumulation be extensive, when it escapes from the antrum into the cavity of the nose, discharging by *one* side. Smaller accumulations of pus find exit in the recumbent position, and attention is called to one antrum as the seat of affection by noting that in the morning pus appears at but *one* nostril. The discharges from purulent nasal catarrh appear upon both sides.

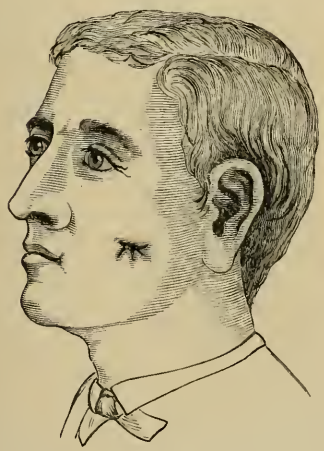
A more common history of antral empyema is the patient's complaint of dull, heavy pains and uneasiness over one side of the face, and an offensive odor, which may not be evident to the operator. High illumination of the mouth by means of the electric mouth-mirror may reveal the presence of fluid in the antrum. Examination of the posterior teeth will show one of them to be pulpless. If such a tooth be extracted, a profuse flow of pus may follow, and a probe may be passed through an alveolus directly into the antrum. Although this is the usual surgical relief, dental conservatism rebels against the immediate condemnation of the offending tooth. Efforts at curing the antral condition through the pulp-canal are well-nigh hopeless—the antrum is entered at some other point. The tooth is treated as any infecting root; is sterilized and filled. The most certain spot of entry to the antrum is about one-quarter inch above the buccal roots of the upper first molar. The part, or the patient, is anæsthetized, and the soft tissues incised or a section removed by means of a tubular knife; a drill or trephine at least one-eighth inch in diameter, driven rapidly, is passed upward, backward, and inward, piercing the thin shell of the antrum at this point. The nozzle of an

atomizer, filled with a 3 per cent. solution of pyrozone, which has been rendered alkaline by sodium dioxid and warmed, is passed into the antrum and the cavity is freely sprayed. A probe is passed into the cavity and an exploration made to detect the presence of any dead bone, which, if found, must be removed, the cavity of entrance being enlarged to permit its removal. The cavity is sprayed about every other day with very dilute, warm Dobell's solution.

In cases of long-standing chronic abscess, with fistula opening at a distance, the fistula may refuse to heal. In that event the tract should be scraped and painted with 20 per cent. silver nitrate solution. If healing does not occur, the offending tooth should be extracted.

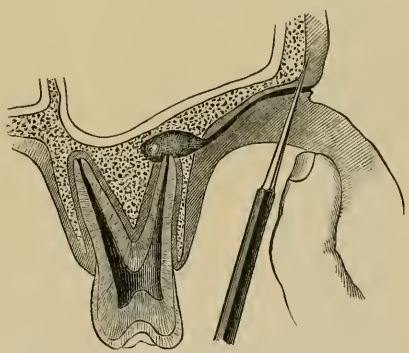
In fistulæ discharging upon the face the formation of scar-tissue may bind the tissue of the cheek tight to the bone. When this occurs beneath the tip of the chin, the scar, after healing, usually resembles a dimple, and calls for no interference. The scar and binding down along the border of the inferior maxilla, or beneath the malar bone in the upper maxilla, may produce deformity calling for remedy (Figs. 326 and 327). Black's operation is to be performed to lessen the

FIG. 326.



Scar caused by alveolar abscess discharging on the face. (Black.)

FIG. 327.



Operation for the remedy of scar on the face caused by alveolar abscess. (Black.)

deformity, for its complete correction is not practicable. A finger placed in the mouth draws the cheek away from the alveolar wall, when the exact position of the cord of attachment is discovered. A tenotome-knife is passed into the tissues, dividing the band of attachment; a long pin is passed through the most depressed portion of the scar, its centre, the long ends of the pin resting upon the face; strips of adhesive plaster laid upon the skin under the head and point of the pin will prevent the latter sinking into the soft tissues. The pin is retained for several days, until the cut in the mouth heals.

Chronic Septic Apical Pericementitis (Non-purulent).

Continued inflammation of a low grade, or what may in reality be continued atonic hyperæmia, may exist in the apical pericementum for long periods without pus-formation.

Fig. 328.

Cause.—The cause of the condition is the presence of decomposing matter about the apex of the root. This is most frequently associated with imperfect root-fillings. After apparent sterilization of canals, their filling has not been complete, so that serum percolates into the canal and in all probability mixes with decomposing tubuli-contents. As noted on p. 389, it requires a longer time and more complete and stronger antisepsis to completely sterilize canals than are usually given. Miller's[1] experiments have shown that the infection of root-tubuli is only for a scant depth (Figs. 328 and 329), and that minute fragments of the pulp itself must be regarded as the offending agents. It is only by way of the pulp-canal that infection need be feared. The depth

Fig. 329.

Fig. 328.—Sector of a cross-section from a diseased root: *a*, cement; *b*, stratum granulosum; *c*, very narrow and finely branched tubules. × 150.

Fig. 329.—Dentin from the root of an abscessed tooth, showing the penetration of cocci to a depth of about $\frac{1}{15}$ mm. ($\frac{1}{250}$ in.); the side *a*–*b* bordered upon the canal. × 1000. (Miller.)

of bacterial penetration into the tubuli will show infection laterally

[1] *Dental Cosmos*, 1890.

through tubuli and cementum to be highly improbable. A chronic inflammation is usually noted in connection with non-carious teeth whose pulps have died in consequence of injury to the apical vessels.

Symptoms and Diagnosis.—The symptoms of this condition are tenderness upon decided pressure or upon percussion; the response may only be elicited by pressure or percussion in one direction. The tooth gives a dull note upon percussion and is usually looser than its neighbors. The red line of the gum extends farther toward the gum-margin than normal—quite to it in some cases. Evidences of a dead pulp are sought; large fillings, the presence of opacity in the tooth, or a markedly different color from that of its neighbors, all point to this; which an examination with the reflected light of an electric mouth-mirror confirms. The tooth by this light is seen to differ in opacity from its neighbors.

An effective method of application of the thermal test, is that suggested by M. L. Rhein: the tooth is isolated and a spray of methyl chlorid is directed against it. A live pulp responds immediately to the intense cold produced; a dead pulp fails to respond.

Examination of the tooth is made to see whether it occludes improperly; if a filling be present, whether the latter show occlusion-marks; excessive occlusion gives rise to symptoms resembling those of chronic septic apical pericementitis. If faults in this direction exist, they are to be corrected and the effect noted. If they do not exist, a septic origin is diagnosed.

Treatment.—The treatment is that of pulp-gangrene: the tooth is sterilized and isolated, and its root-canals entered to the apex and sterilized. The presence of decomposing organic matter is shown by the bubbling which ensues when a 25 per cent. solution of pyrozone is placed in the canals. Canal-fillings are removed even when *apparently* perfect. The condition is common under cotton canal-dressings, the latter usually emitting an offensive odor. Gutta-percha stoppings are warmed by blasts of hot air and removed by fine hooks. Oxychlorid fillings are removed by means of sulfuric acid, which is also applied to all fine canals containing no fillings. Gold, tin, and amalgam root-fillings are practically irremovable. A careful examination is always to be made for extra canals.

There can be no assurance of safety until a fine canal-cleanser can be carried to the apex of the root, until the patient winces. The cleansing is to be accomplished with strong sodium-dioxid solutions. In case of failure to reach the apex, after a prolonged cleansing with sodium dioxid, the canal is washed out with a solution of hydrochloric acid, a zinc electrode inserted in the root, and a cataphoric current applied; the zinc oxychlorid formed is forced into all interstices. This action should not

be prolonged, or the pericementum may become very much irritated by an excessive amount of the zinc salt. Such canals are best filled by first thoroughly drying, then flowing into them the melted paraffin and aristol mixture, and thrusting into the fluid filling a long, slender, metallic point, made hot.

If the pericementitis have been of long standing, the thickening of the membrane will have caused protrusion of the tooth. The tooth should be ground off at its point of occlusion until it occludes with somewhat less force than its neighbors, the therapeutic principle in these cases being that of removing the source of irritation and procuring surgical rest. Indications of favorable results are found in the red gum-line assuming its normal position, tenderness disappearing, and increased tightness of the tooth.

This affection is extremely common about the roots of pulpless teeth, and always signifies more or less enforced disuse of the teeth, and, if uncorrected, their ultimate loss.

CHAPTER XXIII.

NON-SEPTIC GENERAL AND APICAL PERICEMENTITIS.

Apical and general pericemental disturbances of a vascular type and of non-septic origin present in two classes, the acute and the chronic.

For purposes of classification they may be grouped as follows:

- **Acute**
  - Traumatic pericementitis
    - Blows.
    - Wounds.
    - Wedging.
    - Orthodontia.
  - Perforated roots
    - Non-septic.
    - Septic.
  - Chemical agencies — Canal medicaments.
- **Chronic**
  - Perforated roots.
  - Excess of root-filling — Acute also.
  - Excess of crown-filling — Acute also.
  - Resorption of roots.
  - Hypercementosis (exostosis).
- **General Aseptic Pericementitis**
  - Traumatisms.
  - Over-use of teeth — Mal-occlusion.
  - Misuse of teeth — Mal-occlusion.
  - Disuse of teeth — Non-occlusion.
  - Impacted third molars — Non-occlusion.
  - Drug-action.
  - Gout.
  - Scurvy.
  - Syphilis.

Traumatic Pericementitis.

By traumatic pericementitis is meant an inflammation (profound irritation) of the pericementum, the result of mechanical violence. According to the nature and mode of action of the injurious force, the apical or any other part, or all parts, of the pericementum may be affected.

Causes.—The causes of this condition are many and varied; a transient traumatic pericementitis may be excited after arsenical applications to kill the pulp, by forcibly removing the pulp while it still

retains a vital connection at the apex; by forcing the root-filling material beyond the apex of the root, particularly if the material be of an irritating character, such as zinc oxychlorid. The presence of a neutral canal-filling projecting into the pericementum usually causes a continued pericementitis of a lower grade.

The passage of irritating chemical substances used in sterilizing canals may light up a transient pericementitis. Pericementitis following the introduction of sodium-dioxid or sulfuric-acid solutions usually subsides quite promptly; if strong solutions of zinc chlorid or mercuric chlorid have caused the inflammation, it may be more pronounced and of longer duration.

The passage of reamers through some lateral aspect of a root may light up a pericementitis which resists all treatment. These cases, frequently, are infected either at the time of injury or at a later period.

Too violent wedging is always followed by more or less pericementitis of the wedged teeth and their neighbors, more marked when elastic rubber wedges are used.

In correcting irregularities of the teeth, if they be moved too rapidly, are not firmly directed during the operation, or subsequently not firmly maintained in position, pericementitis of a high grade is frequently excited.

The excessive use of the mallet in building down fillings, particularly upon pulpless teeth, may be followed by pericementitis.

The biting of hard substances, such as nuts and pieces of ice, and notably thread-biting, are prolific sources of traumatic pericementitis.

In addition to these, falls, blows received upon the teeth, and blows received under the chin, bringing the teeth forcibly together, all induce pericementitis, which may vary in degree from a passing soreness to general osteitis and perhaps necrosis.

Prophylaxis.—It will be observed that the majority of these causes are associated with operative dental manipulations, or dental abuse by the patient, and are in a large degree preventable. In treating pulp-canals with such agents as a 50 per cent. solution of sodium dioxid, or a 50 per cent. solution of sulfuric acid, care is taken as the end of the canal is approached not to pump them through the apex, and to neutralize, with an acid in one case, an alkali in the other, at the end of the sitting. Strong solutions of zinc chlorid or mercuric chlorid are kept away from the apices of roots. In case zinc oxychlorid is used as a canal-filling, the apex of the root is gently sealed with a small pellet of cotton containing an antiseptic oil, or, better, a small cone of softened gutta-percha. Canal-fillings are inserted gently and withdrawn as soon as sensitivity is noted; by introducing too large a root-filling mass, air may be imprisoned between the filling and the apex of the root, exciting

a reaction as though the filling itself had been passed through the apical foramen. Teeth are to be supported mechanically while large gold-fillings are being malleted into them. The support is derived through judicious placing of wedges. Severe malleting should not be practised except the pericementum be healthy.

Gradual wedging should supplant the violent wedging with rubber. The latter, when used at all, should be in thin layers, and the wedging completed with tape or wooden wedges lightly applied. Malleting should not be done upon wedged teeth until, after a period of rest, pericementitis has subsided, and unless the teeth are rigidly held during the filling operation.

Orthodontic appliances should be of such types as permit a gradual, steady advance in tooth-movement; and when the irregularity is corrected the tooth should be maintained firmly in its new position until the surrounding tissues hold it firmly.

Patients should be warned against the evil effects of thread-biting and biting hard substances.

Symptoms and Diagnosis.—The general symptoms are soreness and looseness of the tooth, together with varying degrees of vascular disturbance of the gum overlying the tooth. A diagnosis may usually be made by obtaining a history of the case, by excluding septic influences as probable causes, and by the disappearance of the disorder upon applying treatment indicated in traumatic injuries.

Clinical History.—Cases due to the passage of irritating chemical substances through the root-apex subside, as a rule, after a period of a few days. Cases due to the presence of a protruding root-filling usually become chronic, and secondary pathological processes arise. Cases due to wedging subside, as a rule, after a period depending upon the severity of the wedging and peculiarities of the individual. Permanent injury may be done to pericementum from such causes, and a predisposition to degenerative changes be established. The pericementum may become permanently debilitated and perfect tissue-regeneration or organization be prevented in tooth-regulating, if the teeth are permitted undue mobility during regulating or not firmly splinted afterward. In cases due to direct and sudden violence to the pericementum, blows upon the teeth, and the crushing of hard bodies, etc., the acute symptoms may disappear after a period governed by the degree of violence and of tissue-reaction. In these cases, however, and in those of thread-biting, strangulation or thrombosis of the pulp-vessels may occur in consequence of the pericementitis, and at later periods pulp-decomposition may occur and give rise to the septic forms of pericementitis.

Pathology and Morbid Anatomy.—In cases caused by the action of irritating chemicals, the latter cause destruction of an amount of

tissue, and the vascular system and cells react to remove the dead tissue. The amount of tissue-death depends directly upon the amount of the chemical present, the vascular reaction being in correspondence. Presumably the pericementitis persists in some degree until the foreign (dead) material is removed by natural processes.

An excess of root-filling material introduces a foreign body into a sensitive vascular tissue, and presumably the changes noted as due to the presence of foreign bodies in other parts occur—an attempt at removal by phagocytes and subsequent encystment (see Resorption of Roots and Hypercementosis).

In cases due to traumatism, such as violent wedging, rapid movement in regulating, overmalleting, blows, thread-, ice-, and nut-biting, etc., the condition is surgically one of bruise.

The phenomena of active inflammation make their appearance to an extent governed by the degree of violence—exudation, swelling, redness, and pain ; fibrinous and corpuscular exudations occur, and later a reorganization of tissue occurs, in some cases a degeneration, depending upon the completeness with which the indicated therapeusis is applied and upon the vitality of the patient.

Traumatic pericementitis in high degree in the young may be recovered from ; but in the middle-aged and aged it may give rise to a series of degenerative changes which only end with the loss of the tooth.

Cases due to perforation of the root and wounding of the pericementum, after the acute symptoms have passed, commonly assume an irritative and chronic type, the soft tissues included in the perforation being in a state analogous to an ulcer. Many of these cases become infected owing to the difficulty of completely sterilizing the apical portion of the canal which lies beyond them.

Treatment.—If the cause of the condition be still in action, it is to be removed or neutralized. In all cases due to violence the treatment is that adapted to injury ; first, surgical rest of the pericementum. This may be accomplished in two ways ; either by preventing the tooth striking its antagonists, or holding it so rigidly that it cannot move if it does meet them. As a preliminary measure the tooth is gently but firmly lashed to its neighbors by means of ligatures so that it is rigidly held. A swaged cap is either fitted to a neighboring tooth, or the antagonizing teeth are ground away until they fail to strike the injured tooth ; the first method is to be preferred.

In cases involving several teeth, such as all of the incisors, two metallic plates are quickly swaged to cover the posterior teeth and they are cemented in position.

Cases due to mild injury may disappear after painting the overlying gum and surrounding territory with tincture of iodin.

A mouth-wash which affords marked relief in many cases is extract of hamamelis, used in one-half strength several times a day.

Acute pericementitis due to the passing of a canal-reamer into some lateral aspect of the pericementum may occur once in the history of a practitioner; it never should twice. Reaming should be done with such care and deliberation that the operator is certain of the direction taken by the reamer, and he should be prepared to cease reaming as soon as canal curvature is felt. Before a reamer removes the last portion of cementum, separating the instrument from the pericementum, sensitivity is announced by the patient, and the reaming should cease at once. Before reaming any canal, its length and direction should be recorded by measuring upon a soft, fine broach. Should sensation and bleeding occur before this end is attained, the root has been perforated. Sterilization of the canals has presumably preceded the reaming operation; if not, the difficulty is increased owing to the impracticability of thoroughly sterilizing the portion of the canal beyond the perforation. The canal is syringed freely with a styptic antiseptic; for this purpose nothing is better than phenol sodique. As soon as bleeding ceases, the canal is to be filled with some unirritating material which can be placed without exercising pressure; chloro-percha or the paraffin mixture meets the indication. Either filling is flowed into the canal until it is full, and a central core, a cone of gutta-percha, is gently inserted in the fluid filling.

If evidences of persistent pericementitis are noted about the root after this operation, it may be inferred that either the portion of canal beyond the perforation is unsterilized or unfilled, or that the pericementum protests against the presence of the root-filling. Guilford[1] has advised and practised successfully in these cases amputation of the portion of the root beyond the perforation.

Chronic Apical Pericementitis (Non-septic).

Definition.—A non-infective condition produced by the action of a constant irritant about the apex of a tooth-root.

Causes.—Many of the causes of acute inflammation, if of less intensity and continuous, or frequently repeated, give rise to the chronic condition. Prominent among these is the thread-biting habit. Another frequent cause is an overfull filling; a filling projects in such manner that it receives an excessive impact during mastication. A projecting root-filling of any substance, causes continued irritation of the pericementum into which it projects. An analogous condition may exist in perforated roots which have been filled.

Symptoms.—The symptoms of the condition are redness of the

[1] *Proc. Academy of Stomatology*, 1897.

overlying gum, tenderness upon percussion, and some degree of looseness of the tooth.

Effects.—The effects of this condition may vary according to the extent of the irritation and its duration. The irritation may in point of effect be but a continued stimulation of slight or high degree, in which event the effects of stimulation follow—*i. e.*, prolonged arterial hyperæmia and its results. The condition may be one of over-stimulation or irritation proper, with corresponding results. There is much evidence to show that the degree and quality of irritation vary. Inflammatory degeneration may occur—*i. e.*, a formation of embryonic tissue which fails of complete organization, or degenerates. The debilitated part may become infected.

Diagnosis and Prognosis.—The diagnosis of the condition itself is usually made without difficulty, but its nature and causes may at times be very obscure or difficult of detection. It is observed whether some part of a filling or of the tooth itself receives undue impact in mastication; if so, the redundant substance is ground away, and the effect noted. Look well to the incisors and note whether they occlude correctly. A history of the case, when obtainable, is of first importance. Knowledge of the nature and thoroughness of the canal-filling, the medicaments used, etc., is a valuable guide. The discovery of the cause of the condition can only be made by exclusion; possibly existing causes are excluded one by one, in the order of their probability.

The prognosis of the case will depend upon the completeness with which the exciting cause of the condition can be removed, and the character of the pathological changes which have occurred.

Treatment.—The treatment of the condition consists in the removal of the cause, and giving surgical rest. When this is done, provided no secondary pathological processes have arisen, the pericementum recovers. The occurrence of this condition, due to traumatism about unfilled roots, is always a danger-signal. The pulp should be carefully examined to determine its vitality. Pulps frequently die of thrombosis arising from repeated traumatism of the apical pericementum, and the conditions then existing, dead material in proximity to tissues in a state of hyperæmia, invite complicated infections.

Hypercementosis.

Definition.—By hypercementosis is meant a secondary deposit, or an increase of volume of the cementum of a tooth beyond the normal limit. It may be circumscribed or diffuse.

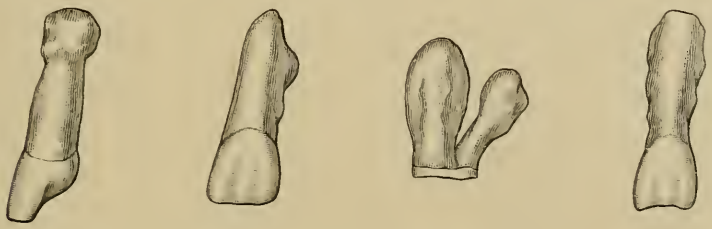
Causes.—Its occurrence is frequently associated with continued non-septic irritation of the pericementum; its causes, therefore, may be any of those described in connection with chronic non-septic pericementitis.

It is found associated with other conditions beside traumatic pericementitis, which conditions will be discussed later. In other cases no direct causes can be ascribed to it; but possibilities in this direction will become more evident from a survey of its probable pathology. In general terms, its cause may be described as a localized or diffused hyperæmia of the pericementum.

Pathology and Morbid Anatomy.—The normal mode of formation and the history of cemental development must be recalled to make pathological formations intelligible. The cementum is deposited as subperiosteal bone in successive layers, beginning before dentinal root-formation is complete.

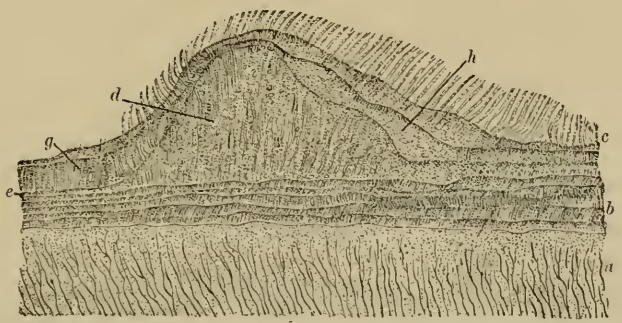
For some time after eruption of the teeth the cementum consists of but few lamellæ of deposit. It differs from bone in that its corpuscles,

FIG. 330.



imprisoned cementoblasts, are irregularly distributed. The cementum is deposited throughout life, so that the teeth-roots of aged persons are

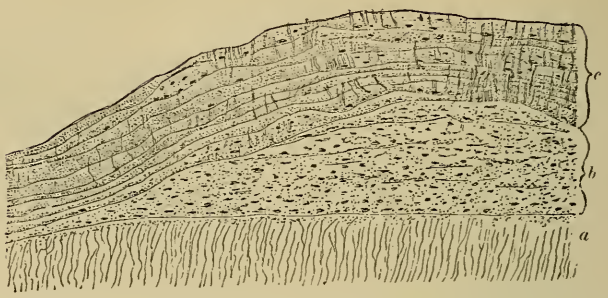
FIG. 331.



Hypertrophy of the cementum on the side of a root of a lower molar near the neck of the tooth of a man: *a*, dentin; *b*, cementum; *c*, fibres of peridental membrane; from *b* to *c* the cementum is normal and the incremental lines fairly regular, but at *d* one of the lamellæ is greatly thickened; at *e* this lamella is seen to be about equal in thickness with the others. The next two lamellæ are thin over the greatest prominence, but one is much thickened at *g*, and both at *h*. These latter seem to partially fill the valleys which were occasioned by the first irregular growth. From a lengthwise section. (Black.)

covered with thick layers of cementum. The maximum of deposition is noted about the apices of the roots, the deposits gradually merging into

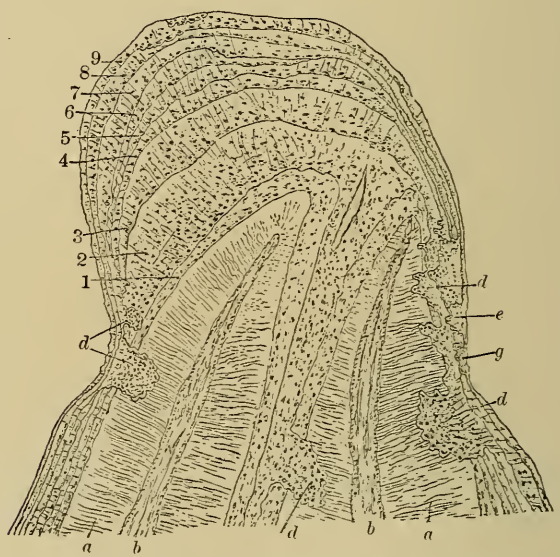
FIG. 332.



Hypertrophy from root of a cuspid in a man, in which the irregularity is confined to the first lamellæ: *a*, dentin; *b*, thickened first lamella; *c*, subsequent lamellæ, which are seen to be fairly regular. (Black.)

the thin lamella of the neck. The deposits occur in successive layers or lamellæ. The formative activity of the cementum, therefore,

FIG. 333.



Apex of root of an upper bicuspid tooth with irregularly developed cementum: *a*, *a*, dentin; *b*, *b*, pulp-canals. The lamellæ of cementum are marked 1, 2, 3, etc.; *d*, *d*, *d*, absorption-areas that have been refilled with cementum. It will be seen that the apices of the roots were originally separate, but became fused with the deposit of the second lamella of cementum, and that in this the regular growth began and was most pronounced. It has continued through the subsequent lamellæ, but in less degree. It will also be noticed that the absorption-areas, *d*, *d*, *d*, have proceeded from certain lamellæ. That between the roots has broken through the first lamella and penetrated the dentin, and has been filled with the deposit of a second lamella. Other of the absorptions have proceeded from lamellæ which can be readily made out. The small points, *e*, seem to have been filled with the deposit of the last layer of the cementum, while others have one, two, or more layers covering them. (Black.)

normally persists to old age, and, like any other function, is susceptible of alterations. It is difficult, therefore, to state exactly where

pathological hypertrophy begins, and physiological new formation ends. Nodular and irregular forms arising from the general surface are clearly of abnormal type. They exist as distinct nodular projections upon some lateral aspect of the pericementum, as a globular mass at the apex of the root, or generally diffused over a greater or less surface of the root (Fig. 330). Each of these probably arises from different causes. Figs. 331, 332, and 333, exhibit the histological characters of the new growth. Outlined portions of the pericementum are seen to have exercised their cement-forming function and caused deposits of successive lamellæ of cementum, in which the cement-corpuscles are irregularly distributed as in normal cementum. Areas are seen where portions of pre-existing cementum have undergone resorption, and where redeposition of cementum has occurred—two distinct vital processes. It is evident that as a result of some irritating influence an outlined portion of the pericementum has been the seat of stimulation; its vital activities have been increased, and the energy expended in formative activity of the cementoblasts. This, it will be observed, is in other parts the outcome of continued arterial hyperæmia and increased functional activity, commonly the consequence of an overwork which is followed by periods of rest. The inference that some spot of mal-occlusion of a tooth, acting as a periodical irritant, is a probable causative agent, is clear. Drawing an analogy from other parts, the altered physiology concerned in hypercementosis is a mild periodical irritation of a more or less localized portion of the pericementum.

It is seen, therefore, how faulty occlusion at some point of a tooth-crown may cause overstraining of the pericementum at a point opposite to that of faulty impact. Also, how a root-filling, such as a projecting cone of gutta-percha, may excite reaction, and at points of pericementum distant from the apex, a constant mild irritation (by continuity) exist in degree sufficient to produce hypertrophy. Again, in other directions, any cause capable of exciting irritation of the pericementum at or near the neck of a tooth, may cause a vascular reaction of milder degree in a more distant part of the pericementum, and thus hypertrophy occur. Such causes are found in the irritation induced by the edges of projecting fillings, overlapping of cavity-margins by the gum, or the presence of deposits of salivary calculi; all of these are in the nature of foreign bodies, and act as mechanical irritants. Hypercementosis is a possibility in any case of chronic pericemental irritation; it represents a degree of irritation, not any specific isolated causes.

Symptoms and Diagnosis.—As the hypertrophy is a result of certain conditions, the symptoms when discoverable are those of the causative conditions. That hypercementosis has symptoms of its own, has never been demonstrated, although a number of symptoms may be

associated with the condition and disappear with the extraction of the tooth. The symptoms are so vague that none of them can be said to be pathognomonic of hypercementosis. The diagnosis of this condition is usually made post-mortem; painful sensations have been experienced, the tooth has been extracted, and the hypertrophy seen—the symptoms disappeared with the loss of the tooth. It is purely a "*post hoc propter hoc*" diagnosis. The *x*-ray, however, furnishes a certain means of diagnosis.

Regarding the pathology of the disease, it is evident that the symptoms will be those of a long-continued mild pericementitis. The tooth or teeth are very slightly sensitive upon percussion. The gum-color may be unaffected; the tooth may not be loosened. The patient complains of a disposition to bite hard upon the particular tooth—to grind upon it, if it be a posterior tooth. These symptoms pointing to pericemental hyperæmia are not necessarily accompanied by hypercementosis, but when such teeth are extracted overgrowth of the cementum is frequently found. In many cases treatment directed to the relief of the hyperæmia causes all symptoms to disappear; that is, if hypercementosis has occurred, it has caused no symptoms after its exciting cause has been removed.

Flagg records[1] cases where neuralgias of the trigeminus, painful functional disturbances of the eye and ear, etc., have been relieved by removing tooth-roots or teeth which were the seat of hypercementosis (see Chapter XXVIII.). The diagnosis of pain about the teeth or head, due to cemental hypertrophy, can only be made by exclusion. Local and general causes of eye and ear diseases having been excluded, the teeth should be examined for sources of irritation. When these exist, they are usually found associated with pulp-disturbance. If pulp-disease can be positively excluded, the possibility of pericemental irritation should be taken into account. If any one tooth show a reaction differing from its fellows, exhibiting pericemental irritability, particularly if upon percussion any increase of the reflex pain be noted, the tooth should be extracted, as the possible cause of the neuralgia. If the neuralgia disappear, it may be fairly inferred that its origin was dental. Entire dentures have been extracted, tooth by tooth, in the vain endeavor to cure a neuralgia about the head. The direct diagnosis of hypercementosis by symptoms is, therefore, very uncertain, and its determination as the causative condition of reflex neuralgia still more so. It is only by use of the *x*-ray that the existence or non-existence of hypercementosis can be positively determined without extraction.

Treatment.—Any tooth which shows evidence of chronic apical or circumscribed pericementitis, even after all discoverable causes of such

[1] *Dental Cosmos*, 1878.

a condition are removed, is usually condemned sooner or later to the forceps. Hypercementosis is a possibility, even a probability, and its causes, whatever they be, are still in operation. It is evident, therefore, that the longer the condition persists the greater will be the mechanical difficulty in extracting. The extraction, difficult though it be, must be complete, or relief from reflex disturbances cannot be hoped for.

(Other phases of hypercementosis will be discussed in connection with general aseptic pericementitis.)

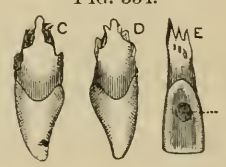
Resorption of the Roots of Permanent Teeth.

Definition.—By resorption of the roots of permanent teeth is meant a condition analogous to that observed upon the roots of temporary teeth prior to the eruption of the permanent teeth.

Pathology and Morbid Anatomy.—Both resorption of cementum and its redeposition occur in teeth as physiological processes; at some aspect of the cementum the tissue becomes hollowed out, and later filled in by new cementum. Resorption of tissue throughout the body is accomplished by means of multinucleated cells. At some part to be physiologically resorbed these cells make their appearance in contact with the tissue to be removed, and it gradually disappears, the layer of multinucleated cells constantly occupying the excavated territory.

If a foreign (aseptic) body be introduced into living tissues, it becomes surrounded by these cells, which in some cases effect its removal; in others, failing to remove the foreign body, connective tissue forms about it and encysts it; encystment may occur after partial removal by giant cells. It has been observed that under conditions of irritation about the necks of teeth, such as those produced by the presence of foreign bodies, that the hard dental tissues may become excavated and deep depressions form. Teeth whose roots have undergone resorption present these conditions: any amount of the apical portion of the root has disappeared (Fig. 334), and if the socket from which it has been extracted be examined, it will be found filled with a mass of soft tissue, resorption of cementum, dentin, and alveolar walls having occurred.

Fig. 334.



Teeth which have been implanted, replanted, or transplanted, about which union more or less perfect has taken place (provided always that suppuration has not occurred), if subsequently extracted, are seen to have been the seat of tissue-resorption, showing excavations into the cementum and dentin. The explanation of these cases is, no doubt, that of the disposal of asep-

tic foreign bodies. Teeth inserted into sockets formed from them, returned to their original alveoli after extraction, or taken from one alveolus and placed in another, are of the nature of foreign bodies, but least of all so, when replanted. If alveolus and tooth be rendered aseptic, the insertion of the tooth is followed by the phenomena of mild inflammation; exudation, fibrinous and corpuscular, occurs, and the tissues endeavor to rid themselves of an intruding body; the inflammatory reaction soon subsides, and giant multinucleated cells attack the tooth-root and endeavor to remove it by solution; this they accomplish in part and in spots; then a tolerance is established and connective tissue organizes about the root; later, more complete regeneration is represented in formation of bone. It is understood that in the primary inflammatory action some portion of the alveolar wall undergoes transformation into embryonic tissue. It will be inferred, therefore, that whenever resorption of roots occurs it indicates a degree of irritation in the surrounding vital tissues probably in excess of that producing local hypertrophy. As the process occurs most commonly in middle age, it may also be regarded as a modified and hastened expression of alveolar atrophy.

Causes.—The possible causes which may be assigned to this condition are naturally those associated with hypercementosis, differing in degree. However, another element must enter into the matter to determine the peculiar tissue-reaction. For want of a better explanation these may be called peculiarities of the individual.

Some of the cases exhibit no tangible cause. Perhaps the most common of the causes discoverable is a protruding root-filling. This condition has been noted as the probable causative association with the peculiar grade and quality of pericemental reaction involved in resorption. Cases occur, however, where the tooth is non-carious and the pulp is alive, direct evidence of vitality being obtained before and after extraction.

Symptoms.—The tooth may be tender upon percussion, and is nearly always loosened; but unless the resorption have progressed far, the latter may not be observed. Later the loosening is peculiar; the tooth moves, as might be expected, with a shortened radius of movement. The condition may be discovered by accident: evidences of mild pericementitis appear, and the pulp-canal is opened to search for a cause. The pulp may be found alive; if alive, and it is killed, or if it is found dead, broaches pass suddenly into the mass of soft tissue underlying the root. The progressive loosening of the tooth, with a shortened radius of movement, is about the only constant symptom of the condition.

In cases of live pulp this organ may be hyperæmic, so that increased response to heat or cold is felt; this, taken in connection

with the tenderness upon percussion which can usually be elicited, and, with the peculiar loosening of the tooth, is a diagnostic guide.

Flagg[1] states that reflex neuralgias occur in this condition, but that the most constant indication noted by him was a sense of discomfort about the jaws, vaguely associated with some one tooth. The patient was convinced that if the tooth were removed, relief would follow.

In the absence of the peculiar loosening of the tooth, which may not occur until the root is nearly gone, a diagnosis is made by exclusion; the resorption is most commonly discovered by entering the pulp-canal and finding its length much shortened.

If the apparatus be available, the *x*-ray should exhibit the condition with sufficient clearness to furnish an absolute diagnosis.

Treatment.—Whether the condition be discovered or not, every possible source of pericemental irritation should be removed. Usually this involves the entrance to and complete cleansing of the pulp-chamber; when the existence of a mechanical irritant in a protruding root-filling, a broken broach or reamer projecting beyond the apex, is discovered and removed. If pain continue, or neuralgia assignable to no other cause persist after removal of all discoverable sources of pericemental disturbance, the tooth should be extracted.

General Aseptic Pericementitis.

Definition.—By general aseptic pericementitis is meant a vascular disturbance involving all or the greater part of the pericementum, and not due to septic causes. Pericementitis is a misnomer in this connection, for in some of the cases the essential phenomena of inflammation may not be present. The condition may be acute or chronic.

ACUTE VARIETY.

Causes.—The causes of acute general pericementitis are mechanical violence, the irritation of foreign bodies, notably improperly adapted artificial crowns, selective drug-action, and gout.

Symptoms.—The symptoms of the condition are tenderness or pain upon pressure or percussion, loosening of the tooth, with injection of the overlying gum; the swelling, like the other symptoms, is less pronounced than in septic pericementitis.

Clinical History.—Cases already described as due to too violent wedging, abuses in regulating of teeth, are fitly included under this head, as are also many cases due to direct violence, such as blows. When artificial crowns of the barrel variety are driven too far under the gum, their edges may impinge upon the pericementum and give rise to inflammation, which may involve the greater portion of the peri-

[1] Lectures.

cementum. Bristles from tooth-brushes, fragments of tooth-picks, and other material may be driven forcibly into the pericementum and cause inflammation. In all of these cases, beginning at the gum-margin or communicating with the mouth-cavity, septic infection almost certainly follows.

If mercury be administered to patients in large doses for long periods, or in one or more massive doses, or if the patient have an idiosyncrasy to the action of this agent, an irritation of the salivary glands is excited, followed by looseness and soreness of the teeth and swelling of the gums; that is, a general pericementitis and maxillary periostitis arise. Potassium iodid administered in this condition relieves the maxillary periostitis and pericementitis; but the same drug administered in health, or for conditions other than mercurial poisoning, also causes irritation of the pericementum. Pilocarpin has a similar effect, though in much less degree. All of these drugs are partially eliminated by the glandular appendages of the mouth, and during elimination apparently act as local irritants.

Patients who have a gouty heredity, or who are the subjects of active gout, frequently exhibit a tenderness of the entire pericementum of one or more, or sometimes all of the teeth. This pericemental disturbance may be the precursor of an acute outbreak of gout in the metatarso-phalangeal joint.

Scurvy—a very rare disease—is attended by rapid degeneration of the pericementum of the teeth and of the alveolar tissues.

Diagnosis and Prognosis.—The history of these cases is all-important. Has an injury been received, or is the patient aware of the introduction or presence of foreign bodies? Did the inflammation arise immediately after the placing of a barrel crown? A history of drug-administration may be obtained, and the constitutional state produced by drug-administration, or by the presence of waste-matters in the circulating fluids, may be evidenced by a widespread disturbance not having a local explanation.

Cases communicating with the mouth-cavity may become purulent, and the condition then becomes acute septic pericementitis, discharging at the gum-margin, and the origin of the condition be thus obscured. Many of these disorders occur about teeth containing normal pulps, as may be shown by the thermal test.

If the exciting cause of the condition be removable, and degenerative changes be not too pronounced, complete recovery may be hoped for. If the source of irritation continue, degeneration of the pericementum usually persists until the tooth is lost, hastened by the infection, which nearly always follows.

Treatment.—The treatment consists in, first, removing the cause,

which the history of the case usually discloses; secondly, procure rest and reduce the morbid vascular condition. The treatment of traumatic cases has already been discussed. If foreign bodies are present, they should be sought for and removed. If the inflammation have arisen immediately after adjusting a barrel crown, and has persisted, a careful examination should be made for loose particles or projecting masses of hardened cement; if not found, the crown should be removed, the inflammation reduced, and a properly made crown adjusted. Cases due to constitutional causes persist as long as the general irritant is present; if this be a drug, as mercury, its elimination is hastened by means of potassium iodid administered internally, and the local symptoms reduced by means of strong solutions of potassium chlorate. If any of the products of faulty metabolism—uric acid and allied substances—be the offenders, they should be gotten rid of by flushing the kidneys, using uric-acid solvents: lithium salts, piperazin, etc. In any event, the pericementum is to be given a rest through means already described, and mouth-washes of hamamelis, etc., advised.

CHRONIC VARIETY.

Causes.—Cases of chronic general aseptic pericementitis are usually due to either the continuance of some of the causes which gave rise to the acute variety, or to overuse, misuse, or disuse of the teeth; these are termed mal-occlusion and non-occlusion of the teeth—either general overwork, improper work, or an absence of work.

The results of overuse, disuse, and misuse of teeth are more correctly described as degenerations rather than expressions of inflammation, although inflammation, simple or infective, may occur at any time in the histories of the cases. The functional abuses comprised in this subheading, by inducing degenerative changes, furnish areas of lessened resistance, points of determination for the lighting up of the condition imperfectly described as pyorrhœa alveolaris. In still other cases localized areas of cellular debility or even cell-necrosis are found, which become infected by pyogenic organisms, forming defined abscess upon some lateral aspect of a tooth, the pulp of the tooth being alive, and the gingival portion of the pericementum still firmly attached.

Overuse of Teeth.

By overuse of a tooth is meant such a variety of occlusion that the tooth receives a greater stress than its neighbors, or than it is designed to bear. The stress may be received in the normal direction, but be excessive in amount. The most prominent cause of this condition is the loss of one or more other teeth, permitting undue stress to fall upon the neighboring teeth, or, in some cases, on far distant teeth. Too-

prominent artificial crowns, particularly those of the all-gold type, cause a general increase of stress upon the pericementum. Enormous contour-fillings overfull may establish a similar condition. When but few isolated teeth remain in one denture and have antagonists, the teeth are certain to be overworked. Isolated and other teeth, to which are attached clasps of artificial dentures, are in the majority of cases being constantly overstrained.

Pathology.—Like any other functional part which is overworked, the pericementum is first stimulated, causing the vessels to dilate. Soon evidences of overwork appear in a passive dilatation of the pericemental vessels, and atonic hyperæmia is established. The condition passes into one of irritation; the tooth projects, and is loosened; the overlying gum deepens in color; and evidences of venous engorgement are common. The result of the condition is a softening and degeneration of the substance of the pericementum; the alveolar wall is involved in the degeneration, and it melts down—disappears to a greater or less extent. At any stage of the disturbance infection may occur, and the degeneration and destruction of the pericementum be hastened by suppuration, or other secondary degenerations.

The symptoms, diagnosis, and clinical history are involved in the description. The prognosis is the inevitable loss of the tooth if the causes be not removed, in which event, the prognosis is governed by the extent to which the degeneration has proceeded.

Treatment.—The treatment is the removal of the causes and procuring surgical rest until the injured pericementum has recovered. The insertion of carefully made artificial dentures is indicated in those cases of scattered natural teeth having spaces between them. The prosthetic appliance must not be attached to these teeth, nor in its movements should it bear against them. No attempt is made, however, to cause the artificial teeth strike before the natural teeth in the hope of giving surgical rest to these organs. Such attempts always result in failure, as they cause injuries to the tissues upon which the plate and teeth rest, which are more severe than the pericemental disturbance.

Properly adjusted bridge-work frequently does good service in these cases, provided the over-occluding tooth or teeth be first dressed down short of occlusion and are given a period of rest, until the pericementum recovers. The bridge, if carefully planned, may be made to direct and control the stress received by the injured teeth.

Improperly occluding artificial crowns should have this fault corrected, by removing the excess of material or by setting properly made crowns.

Overfull fillings should be reduced to correct proportions and shape.

Teeth which are being strained by clasps should have the latter

removed. If necessary, a new appliance should be made on which clasps are either omitted, or are properly designed for other teeth.

Surgical rest is the only hope of saving the tooth.

Mal-occlusion of the Teeth.

Each tooth of a denture is not only designed to receive a definite amount of force, but to receive it in a particular direction or directions; any excess of this force, or alteration of its direction, is followed by abnormal stimulation of the pericementum (see Chapter VIII.), and by its overstraining. The effects following a general increase of stress have been considered under the previous heading. By mal-occlusion is here meant, the constant reception of stress by the pericementum in directions to which it is unaccustomed, or are not in accordance with the anatomical design of the tooth.

Causes.—Original malpositions of the teeth may cause their faulty occlusion. The most prolific source of the condition is, however, altered occlusion due to those changes of position of the teeth which follow upon the loss of adjoining teeth.

Artificial crowns which do not occlude in correspondence with the other teeth are a common cause. Improperly formed fillings are another cause.

The shifting of positions of the teeth, in consequence of pathological changes occurring in or about the pericementum, cause the crowns of teeth to occlude improperly.

Pathology.—The conditions established are either those of overuse or of disuse. A typical example of this condition is that of a lower second molar which has gradually tilted forward in consequence of the loss of the first molar; or a central incisor which has altered its position in consequence of secondary formations in or about the pericementum, a common precursor of phagedenic pericementitis. Some portion of the tooth, an edge, which before did not occlude with an antagonizing tooth, is brought into occlusion; if the occlusion be not unduly forcible, no immediate degenerative changes are evident. If the occlusion be excessive, the pericementum is not uniformly affected, but the greatest stress is brought to bear upon some lateral aspect of the structure. It responds in the degree of the overwork, and degenerative changes occur, which, if the active causes be not removed, gradually spread to other portions of the pericementum, and the phenomena noted in connection with overuse occur, but are not so general in distribution. The tooth becomes more movable in one or more directions—*i. e.*, is loosened; it may develop some degree of tenderness upon percussion, and the gum-color toward the affected side deepens, although it may remain normal in other parts. As in the previous cases, infection may—indeed, is

likely to—occur, and suppurative processes may hasten the pericemental destruction. In some cases the pericementum may degenerate and be destroyed about one root of a multirooted tooth, and remain about the other.

Diagnosis.—In all malposed teeth a careful examination should be made of their mode of occlusion. If the tooth exhibit tenderness and looseness, mal-occlusion is almost a certainty; it only remains to determine its direction.

The spots of faulty occlusion may be determined by placing a strip of carbon paper (articulating paper) over the tips of the antagonizing teeth and having the patient bite; the spots of contact should then be ground away until the tooth is slightly short of direct occlusion. Fresh strips of paper are used, and the jaws moved laterally, as in mastication, to note other points of contact; these should also be ground away. When several teeth are affected to any extent, whether by a primary shifting of position, without evident vascular symptoms, or infection have occurred, and pyorrhœa alveolaris is established, it may be advisable to make accurate models of both dentures, mount them in a Bonwill articulator, and carefully note the points of undue contact. If tin models be made, the points of mal-occlusion may be filed away until the occlusion is normal. The positions of the filed spots on the models furnish infallible guides for grinding the natural teeth.

The study of these conditions of tooth-usage should be made parallel with a study of the conditions included under the generic title of pyorrhœa alveolaris.

Prognosis.—If the condition be not corrected every time occasion requires, the degeneration progresses until the tooth is lost. While there are, no doubt, several other factors which act as predisposing causes of the ultimate atrophy or necrosis of the pericementum—pyorrhœa alveolaris, phagedenic pericementitis, and gouty pericementitis—yet faulty, excessive, or non-usage of the pericementum must take first rank as local predisposing causes.

Treatment.—The principle of treatment is the removal of active causes, implying correction of the occlusion; the removal of dead tissue, including tooth-roots which are entirely denuded of pericementum; and securing a period of surgical rest. The correction of the occlusion has been described under the head of diagnosis. Subsequent corrections are usually required at intervals.

Artificial crowns need special scrutiny to see that the occlusal surfaces are properly restored.

When atrophy and death of portions of the pericementum have occurred, conditions are established which are described in subsequent

chapters, where their clinical histories, treatment, etc., are also discussed.

Disuse of Teeth.

Definition.—By disuse of teeth is meant a degree of usage less than the amount, the forms, and structure of the teeth and contiguous parts fit them for. The disuse may be absolute or relative; teeth may not occlude at all, owing to the loss of antagonists or to extremely irregular positions.

PARTIAL DISUSE.

Causes and Clinical History.—The meaning of relative disuse needs exhaustive inquiry. Black's[1] experiments have shown that the strength of the muscles of mastication, and the amount of stress the pericementum will bear, differ with the individual (see Chapter VIII.). One person taking a morsel of food, such as a fragment of meat, between the teeth may crush it flat with one closure of the jaws; another will require continued mastication to reduce it. The amount of exercise the teeth (the pericementum) receive in the first individual is much greater than with the second. Moreover, the teeth and alveolar process of the first are of a type called highly organized. If in either or any case, soft food be substituted for that requiring strong mastication, the functional activity of the pericementi is lessened because of lack of exercise. Naturally the effects are in the degree of lessened exercise, all other factors being equal; and this amount is relatively least where the teeth are of a type designed for hard usage.

Pathology and Morbid Anatomy.—Lack of exercise is necessarily followed by atony; and if it occur in a part accustomed to vigorous exercise, degenerative changes proportioned to the degree of disuse are sequelæ. Atony, debility, etc., are followed by the less pronounced degenerations, and result in atrophy. In all cases, whether relative or absolute disuse, the bloodvessels of the pericementum share in the atony, and a passive dilatation or hyperæmia results; lacking in varying degree the stimulus derived from mastication (exercise), and the adjunctive circulatory force exerted thereby, the vascular current becomes sluggish, and areas of lessened resistance are formed. The vascular conditions are the antecedents of degeneration. The clinical importance of the condition at the stage described, is that it frequently precedes persistent atrophic conditions of the pericementum—*i. e.*, phagedenic pericementitis. In gouty patients it establishes a weak joint, which may be the point of deposit of gouty poison. The gum-tissues also may become debilitated, owing to the absence of their normal stimuli, and passive hyperæmias result. Other evils follow; food-débris collects

[1] *Dental Cosmos*, 1895.

in unusual amounts, and abnormal fermentations occur, producing gingivitis (which see).

Diagnosis and Prognosis.—A diagnosis of disuse (relative) is usually made out by inquiring as to the food-habit of individuals. It is excessively common in civilized communities, particularly among the well-to-do, and is of almost constant occurrence in gourmands.

Treatment.—Patients should have pointed out to them the results of insufficient mastication, together with the evils of faulty oral hygiene. Every effort should be made, by the use of antiseptic and mildly astringent mouth-washes, to forestall the common sequel of these conditions, progressive degenerations and loss of the teeth. These and similar conditions are particularly to be feared in the degenerative periods of early and late middle-age. It is between the ages of thirty and fifty years that ill-consequences are most to be feared from acquired debility of the pericementum.

ABSOLUTE DISUSE.

Definition.—Teeth which perform no work directly in mastication, or indirectly by serving as abutments for a bridge-piece, may be said to be in a condition of absolute disuse.

Results.—A tooth or root whose pericementum receives no stimulus becomes relatively a foreign body to the organism. It is a useless part, and the body attempts to cast it out. Perhaps these phrases are insufficiently exact; however, a disused tooth is lost through a series of pathological changes. Teeth which perform no work may be retained in the mouths of young children and young adults for long periods without marked changes occurring in their vital connections, but during the degenerative period of life they are usually lost with a degree of rapidity differing in individuals.

The pericemental condition of passive hyperæmia following upon relative disuse of the teeth has been described; the condition following upon absolute disuse differs in that the pericementum receives no exercise whatever. The clinical history of these cases is that of a progressive extrusion of the tooth; it projects beyond its fellows in increasing degree. The borders of the alveolar process recede, but usually to less extent than the tooth protrudes or is extruded. The tooth becomes progressively looser, until in its latest stages a portion, which may be one-half of its root-length, is attached to the jaw through the medium of a mass of soft tissue alone; all true alveolar connection has disappeared. After extraction or complete extrusion, the root of the tooth is seen to be devoid of pericementum except at the apex of the root. The alveolar process has undergone limited atrophy, although in some cases its outer walls may be thickened.

Pathology.—The passive hyperæmia has apparently led to swelling and degeneration, with subsequent atrophy of the pericementum, and the normal atrophic changes which occur in the alveolar process have become hastened and quickened. These cases frequently become complicated by infections, when the tooth-loosening becomes pronounced. The pulp-vessels are cut off and the dead pulp-tissue furnishes a soil for micro-organisms, whose poisons hasten degeneration of the tissues in the abnormal alveolus. Suppuration may occur—*i. e.*, abscess form. Through this process the jaws cast out crownless roots; in these the local alveolar atrophy may be complete before there is external evidence of it. The danger of infection is always great in these cases. Some degree of infection, no doubt, exists in all of them, which serves to explain the increased rapidity of the degenerations.

Prognosis.—If teeth can be directly or indirectly brought into use, so that their pericementi receive exercise, the cases may recover, provided the atrophic changes are not very pronounced; in which event the atrophy proceeds, although more slowly. Teeth crowned or made abutments for bridges, after degenerative changes have become established—*i. e.*, when the normal pericementum has been replaced by a thickened mass of partially organized connective tissue—usually become progressively looser; the alveolar atrophy proceeds until all attachment is lost. Utilized early, the teeth may be saved. The results are better if the teeth or roots be utilized before the age of thirty than at later ages.

Treatment.—The treatment, as might be inferred from the foregoing statements, consists in bringing the teeth into use, if the degeneration have not proceeded too far. Later, extraction is inevitable. The operation, when determined upon, should not be delayed, for not only are bacterial growths invited about the loosened tooth, but the soft tissues are frequently increased in volume, if extraction be delayed until complete local atrophy of the alveolar walls has taken place, a soft and spongy mass remains, which interferes with the comfortable wearing of prosthetic appliances in the future.

CHAPTER XXIV.

PERICEMENTAL DISEASES BEGINNING AT THE GUM-MARGIN.

Nearly all the degenerations of the pericementum which begin at the gum-margin are sooner or later accompanied by suppurative processes, which give a generic name to these conditions, viz., pyorrhœa alveolaris. Under this head dental writers have included several disease-processes which should be clearly differentiated from one another. In general terms, these diseases are characterized by an inflammation originating about the gum-margin, and followed by a progressive degeneration and atrophy of the pericementum and of the alveolar walls. In the areas of pericemental atrophy and death, progressive deposits of calculi take place, and infection of the disease-territory by pyogenic organisms is the rule. Their characteristics, therefore, are loss of pericementum in any direction, forming pockets in which calculi deposit, and from which pus exudes or may be pressed. The primary cause of the atrophy, pericemental necrosis, calculi, and infection are so clearly associated with a primary affection of the gums about the necks of the teeth, that a critical examination of the causes, clinical history, and pathology of inflammation of the gum-margin is a necessary preliminary to the study of the later degenerations.

Gingivitis.

Definition.—The term gingivitis, as at present understood, applies to an inflammation confined to the margins of the gum about the necks of the teeth, in contradistinction to general inflammation of the gums, called ulitis. Fault may be found with this distinction as not being warranted by etymology or dictionary definitions, so that perhaps "marginal gingivitis" would be a more correct term.

The causes of marginal gingivitis are local and general, which may be subdivided into predisposing and exciting. Any conditions, general or local, which reduce the vitality of the tissues forming the gum-margins will cause a predisposition to local disease, when exciting causes may become operative which before were inoperative. It is still an open question whether this is the mode of action of what are known as the general or constitutional causes of marginal gingivitis. Be this as it may—and it is an important question—marginal gingivitis is an

associate of several general disease-states and conditions of faulty metabolism. Rhein[1] found after repeated examinations of hospital patients that "marginal gingivitis was an accompaniment of typhoid fever, tuberculosis, malarial disorders, acute rheumatism, pleurisy, pericarditis, and syphilis, among the acute diseases. Of chronic nutritional diseases, it was commonly observed in cases of gout, diabetes, chronic rheumatism, several forms of nephritis, scurvy, chlorosis, anæmia, leukæmia, and pregnancy. Also in disorders of the central nervous system and following the administration of mercury, lead, and iodin."

In some of these disorders, notably typhoid fever and other acute diseases, diabetes, pregnancy, and disorders of the nervous system, local causes are sufficient to account for the gingivitis, and may completely mask connection with the constitutional causes. The connection with gout and other arthritic diseases, Bright's disease, anæmic disorders, and tuberculosis is clear and undoubted. Rhein states that the gingivitis produced by any of the causes named has distinctive features which may even serve as diagnostic signs of the nature of the general malady. To render the problem less complex, and discover more general causes, the nature of the tissue-changes induced by these diseases must be examined. Most of them, it will be observed, may be classified as diseases of suboxidation; diseases in which the oxidizing element of the body—hæmoglobin—is in reduced amount; where products of insufficient oxidation are formed or retained, and where probably faulty oxidation is the result of cell-disorders; oxygen in insufficient amount; cells unable to oxidize and deoxidize; oxidizable material in undue amount or of imperfect character; and, lastly, a retention of waste-products of tissue- and perhaps of food-metabolism in the circulating fluids. As in all nutritional disturbances, degenerative disorders most affect parts peripheral to the circulation—the parts become debilitated.

The local causes of gingivitis, some of them probably necessary in all cases to the occurrence of the disorder, are more tangible. The local causes may be divided into predisposing and exciting.

The local predisposing causes are lack of exercise, the gums not receiving the usual friction and buffeting by food-masses during mastication, and permitting the food-masses to remain in contact with the gums, where the products of their decomposition act as irritants. The local exciting causes are both mechanical and chemical, and act vigorously in the degree that local and general predisposing causes exist.

The mechanical causes are the presence of foreign bodies, such as deposits of salivary calculi resting upon the gum or beneath the gum-margin; fillings projecting beyond cavity-margins; gum overhanging cavity-margins; projecting edges of artificial crowns; tooth-brush

[1] *Dental Cosmos*, 1894.

bristles; fragments of bone tooth-picks; improper contact of the edges of prosthetic plates or appliances about the necks of the teeth; injuries inflicted by rubber-dam clamps, ligatures, wedges, etc.

The chemical causes are the presence in the gum-tissue of irritant drugs, probably in process of elimination—lead, mercury, iodids; the contact of caustics applied in the treatment of other dental diseases, and chemical poisons generated through the action of mouth bacteria. The latter cause probably complicates all the others.

Symptoms.—The symptoms depend upon the cause. Swelling and increased redness of the gum-margins are constant features. In gingivitis due to any of the constitutional causes named, the affection is general, but the symptoms are more marked in some diseases than others and usually are pronounced in proportion to the neglect of oral hygiene. In some cases there may be only a raised, rounded, softened ridge of gum, of a bright red color; but in other cases, the swollen gum may obscure fully one-half of the tooth-crown, and the softened tissue be purplish in color.

Prognosis.—The prognosis of marginal gingivitis is governed by its causes, the length of time they have been in operation, and the age of the patient. Gingivitis commencing at the degenerative period of life, particularly when conditions exist which lessen the normal resistive power of the pericementum, is certain to establish degenerative changes in the latter structure. Degenerative and atrophic changes of the pericementum and alveolar walls are inevitable in long-continued gingivitis.

If the trouble be purely local—that is, due to local causes alone—it usually subsides promptly upon the removal of the cause. The pericemental and alveolar atrophy which has occurred makes, however, a permanent loss, so that when recovery occurs, the gum-line is seen to have receded beyond its normal line of attachment to the teeth.

Treatment.—The treatment of the condition consists in removing the source of irritation and restoring the normal circulation in the parts. If the source of the disorder be in some underlying constitutional condition, the symptoms may be ameliorated, although not entirely cured, by the correction of the general disorder.

Cases due to mechanical irritation are commonly confined to one or several teeth, rarely to an entire denture, except cases continued in consequence of deposits of scaly calculi beneath the gum-margin. Foreign bodies, such as bristles and fragments of bone, should be removed. Projecting fillings or overhanging crown-margins should be made flush with the general tooth-surface. Salivary calculi should be removed.

Antiseptic mouth-washes should be employed frequently, no matter what the cause. If the gum-tissue be soft and spongy, showing signs

of venous hyperæmia, antiseptic, astringent mouth-washes should be freely used.

℞. Zinc. chlorid,	gr. x;
Aq. menth. pip.,	℥j.—M.

used in spray from an atomizer or as a wash several times a day is an excellent local application, meeting both indications. Preparations containing carbolic acid and allied substances do not appear to act happily in these cases. Prescriptions containing eucalyptus and benzoic acid are to be preferred:

℞. Acid. benzoic.,	3 parts;
Tr. eucalypti,	15 "
Ol. menth. pip.,	1 part;
Alcohol,	100 parts;
Saccharin,	2 " —M. (Miller.)

The above formula diluted one-half is agreeable and efficient.

Listerine, borine, borolyptol, and other preparations of thymol, boric acid, eucalyptus, etc., are all useful when conjoined with the removal of every local and general cause discoverable.

Specific local causes of gingivitis, such as salivary calculi, require special consideration.

Salivary Calculus.

Salivary calculi are hard formations composed of the calcium salts of the saliva which have been deposited or precipitated, and combined in an unknown manner with organic substances, usually mucin.

Occurrence.—They are found upon the surfaces of the teeth, notably in situations opposite the mouths of the salivary glands; in the ducts of the muciparous salivary glands (sublingual and submaxillary), and beneath the margins of the gums.

Varieties.—Clinically three varieties of salivary calculi are recognizable: first, the soft, friable, whitish-yellow deposits found upon the buccal surfaces of the upper molars and upon the lingual surfaces of the lower anterior teeth; second, dark-colored and hard deposits found more frequently in the latter situation, less frequently in the former; third, dark, hard, scaly deposits found first immediately beneath the gum-margin and extending from that point. Deposits upon the teeth have been divided into salivary and sanguinary or serumal, ptyalogenic and hæmatogenic calculi (Peirce), and the scaly deposits named have by some writers been classified as hæmatogenic or serumal, which is incorrect; as stated by Peirce, their origin is ptyalogenic, not hæmatogenic.

Serumal calculi are of two varieties, and of different origin from the ptyalogenic calculi.

Causes.—The causes of deposits of salivary calculi are local and, in some cases at least, general. The causes will be more evident after a study of the conditions under which the saliva deposits its calcium salts.

The secretion of the parotid gland is more watery than that of the sublingual and submaxillary glands; it contains a globulin but no mucin, and contains calcium carbonate, calcium phosphate being present in but minute amount.[1] It contains sufficient carbon dioxid to hold the calcium salts in solution. The secretions of the submaxillary and sublingual glands contain calcium carbonate and calcium phosphate in nearly equal amounts and are rich in mucin; that of the sublingual gland contains the highest percentage of solids, particularly of mucin. The mucous glands of the mouth have a viscid secretion (mucin) and contain some 20 parts per thousand of solids, organic and inorganic (Jacobowitsch). It is, therefore, thicker than the salivary secretion proper. The combined secretion, as found in the mouth in conditions of health, is a transparent, slimy fluid of alkaline reaction, containing epithelial (salivary) cells, and from which carbonic anhydrid may be pumped; sufficient carbonic anhydrid is present to hold the calcium salts in solution. As soon as the fluid is exposed, at rest, in a vessel, the carbonic anhydrid escapes, and the calcium salts, being no longer held in solution, are precipitated, the saliva becoming cloudy.

In perhaps a majority of human mouths, certainly in the great majority of those tested by dental observers, the saliva has an acid reaction. Fermentation, particularly lactic fermentation, is so common in the human mouth as to be almost a characteristic. It is due, no doubt, to the addition of the product of this fermentation, lactic acid, to the general saliva that the fluid acquires an acid reaction. Similar conditions are established in the mouths of animals fed upon cooked starchy foods; carnivorous and herbivorous animals are affected, both domestic, and wild animals in captivity. It has been observed that mineral acids, and among the organic acids acetic acid, have the power of precipitating the mucin. Lactic acid has a similar property,[2] exhibiting particular features. If to a test-tube half-filled with saliva, a few drops or more of a 1 per cent. solution of lactic acid be added, a cloudiness will appear in the solution; shred-like coagula of mucin are formed which slowly agglomerate and rise to the surface of the solution. If the amount of acid be increased, the coagula form more promptly and agglomerate quickly in a distinct mass at or just beneath the surface of the solution. If the coagulum be removed and dried, it is

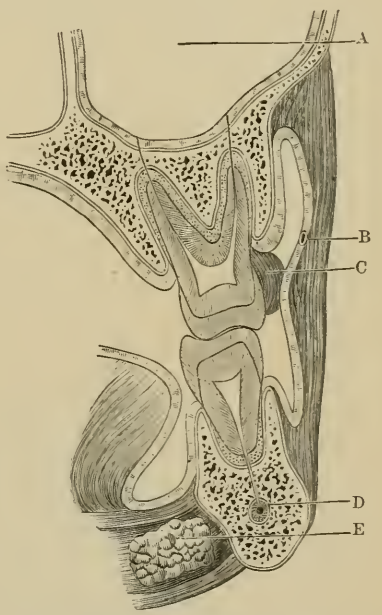
[1] Halliburton, *Chemical Physiology and Pathology*, after Mitscherlich.

[2] Burchard, *Dental Cosmos*, 1895.

found upon analysis to contain calcium salts. The conditions of the formation have been, therefore, a volume of saliva in quiescence becoming acidulated; throughout the solution mucin is being coagulated, and at the same time calcium salts are being thrown down in consequence of the escape of their solvent, carbon dioxid. These salts are entangled in the mucin-coagulum as it agglomerates and rises to the surface. The dried coagula slowly change color, acquiring a greenish-brown hue. Kirk[1] believes that the connection between the calcium salts and the organic substance to be more intimate than a mere cementing together of the calcium particles. Conditions exist somewhat analogous to those under which calco-globulin forms, and he advances the suggestion that salivary calculi may have a family resemblance to calco-globulin. To bring about these conditions it is not necessary that mucin should undergo coagulation, its inspissation is alone sufficient; but some explanation is required of the reasons why calculi are found in selective situations.

The human mouth may be divided into two cavities, a buccal and a lingual; so far as the accumulation of saliva is concerned, the receptacles form but parts of each of these two cavities. A lingual cavity, bounded laterally by the lower alveolar walls, beneath by the floor of the mouth, above and behind by the tongue, is that into which the submaxillary, sublingual, and numerous mucous glands discharge their secretions (Fig. 336). A cavity bounded internally by the buccal alveolar wall and buccal surfaces of the upper molars, externally by the cheek, above by the junction of cheek and gum, below by the edges of the teeth and cheek, is that into which the secretion of the parotid gland is poured (Fig. 335). When the muscular appendages of the mouth, tongue, cheeks, and lips, are at rest these cavities become filled with saliva. The almost constant muscular movements of the structures named keep the fluids in a constant state of agitation; the agitation, flow and interchange of fluids are much increased by active

FIG. 335.

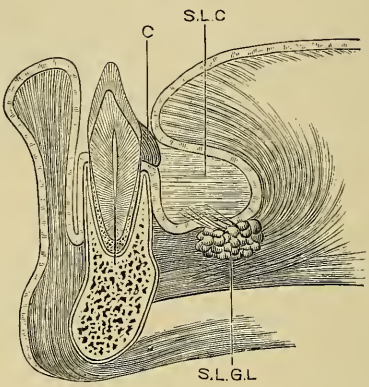


A, maxillary sinus; B, duct of Steno; C, parotid calculus; E, submaxillary gland.

[1] *Dental Cosmos*, 1895.

mastication. Establish now a condition of quiescence, increase the secretion of mucus by irritating mucous glands, increase also fermentations, and new relations are established. The close sympathetic association of the branches of the trigeminus must be borne in mind; irritation of the

FIG. 336.

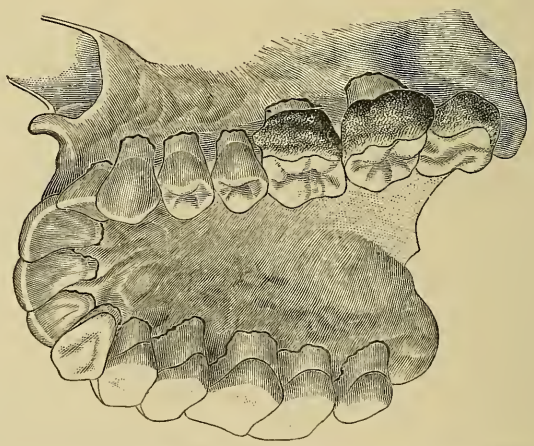


C, calculus; S. L. C., sublingual cavity; S. L. G. L., sublingual gland.

mucous surfaces of the mouth is followed by increased glandular activity. Irritation of exposed dentin, or of an acutely diseased pulp, is followed by increased glandular activity, and if the irritation be prolonged, the character of the secretion is altered.

The increase, or establishment, of these conditions is found in non-mastication; if one or more teeth, especially upper molars, be in

FIG. 337.



Right side, abrasion from over-use; left side, deposits due to stagnation.

continuous disuse, it is usual to find accumulations of calculi upon them (Fig. 337). If catarrhal conditions of the mouth (increased mucous

secretions) occur and persist for long periods, calculi are almost certain to form. As these cases are commonly associated with active oral fermentations, an increased production of acid occurs.

The general composition of calculi is usually given[1] as "calcium phosphate and carbonate admixed with mucus and leptothrix." The secretion of the parotid gland, containing but a trace of calcium phosphate with its calcium carbonate, gives rise to calculi having a corresponding composition; in the calculi upon the buccal faces of upper molars calcium carbonate predominates.

Formation.—Each of the three varieties of salivary calculi exhibits different conditions of formation. It is common, however, to find two varieties combined—*i. e.*, the conditions of formation have been added to one another.

The typical parotid calculus is soft, friable, and whitish-yellow, acquiring density with age. The conditions under which this variety forms appear to be more or less disuse of the teeth of that side. This is well illustrated in Fig. 337. Owing to the loss of the antagonizing teeth of one side, the upper posterior teeth of the same side have fallen into disuse: on the opposite side the teeth have all been worn down by mechanical abrasion. The disused teeth are heavily encrusted with the yellowish friable variety of calculi. The deposit itself probably occurs as follows: more or less disorder of the gum-structures follows upon lack of mastication—*i. e.*, the mucous discharge is increased; in the buccal cavity (Fig. 335) an accumulation of parotid saliva in a state of comparative quiescence takes place; the gaseous carbon dioxid escapes and the calcium carbonate is precipitated; combining with any adhesive matter which may be present, notably the diluted mucous secretion of the local glands, collections are formed which lodge in the interproximal spaces and in the small groove between the gum and tooth. Successive portions of a like character are formed and are added to the original deposit.

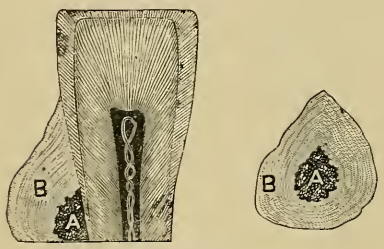
The second variety of calculus, that which deposits first upon the linguo-cervical portions of the lower anterior teeth, and subsequently between the teeth and at the cervico-labial portions, contains a greater amount of calcium phosphate and mucin. The light mucous coagula found in the lingual cavity rise to the surface of the saliva contained in this cavity, and come to rest at the cervico-lingual borders of the lower incisors; the pressure of the tongue, as may readily be tested, tends to drive the coagula or inspissated mucus, which entangle the precipitated calcium salts, between the teeth, moulding them closely about their necks. Subsequent depositions and precipitations occur, which cause accretions to the first deposits. When catarrhal gingivitis is

[1] Vergue, *Du tartare dentaire et de ses concretions*, Thèse, Paris, 1869.

lighted up from any cause, and it is an inevitable result of the presence of these foreign deposits in contact with the gum, it is seen that the calculi change their physical characteristics.

The calculi deposited in the regions of the gingivitis are harder, less in amount, and usually dark green in color. They usually intrude beyond the gum-margin. The swelling incident to the gingivitis causes the formation of a V-shaped depression between the swollen gum-edge and the surface of the tooth. It is in these pockets that the hard, green, closely adherent deposits occur. Similar deposits may be noted beneath the yellow and softer calculi, and if sections of extensive calculi be made, these greenish deposits may be seen scattered through the mass. The greenish deposits, when they occur alone, are usually in the form of very hard scales, closely adherent to the necks of the teeth and lying within the free gum-margin.

FIG. 338.



A, nidus; B, calculus.

Sections of extensive calculi show them to be made up of concentric layers (Fig. 338). Foreign bodies are sometimes entangled in the mass. In some cases extensive salivary deposits are found associated with highly offensive odors; either their presence, or the conditions under which they are formed, appear to invite putrefactive decomposition in the mouth. In the mouths of smokers deposits of carbon are formed upon calculi, giving them a jet-black surface.

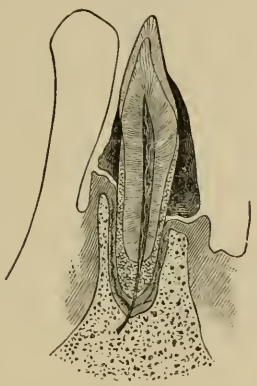
Pathological Effects of Calculi.—The effects of deposits of salivary calculi are immediate and secondary, and their nature is governed largely by the character of the deposits and by the existence or non-existence of predispositions to pericemental degenerations. In some forms they establish immediate predispositions to these degenerations. Their purely local significance and effects must first be considered.

In contact with the mucous membrane, a salivary calculus acts as a local mechanical irritant and excites the reaction noted in connection with other local irritants; the form, consistency, composition, and smoothness, however, represent a milder type of irritant, and naturally their presence is not causative of pronounced inflammatory reaction.

Figs. 339, 340, 341, and 342, represent the relations of deposits of the larger, yellow deposits of calculus upon the lower anterior teeth and upper molars. It is seen that these deposits rest on the gum, and do not insinuate themselves between gum and tooth in such a manner as to sever their attachment. They excite hyperæmia of the gum underlying them, and resorption of the alveolar walls occurs, beginning at the margin; the pericementum and alveolar periosteum recede with the shrinking

alveolar wall, gradually lessening the attachment of the tooth. Successive deposits of calculi occur, which encroach upon the denuded tooth-

Fig. 339.



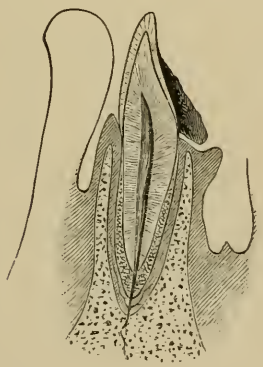
Section of a lower incisor, with a large deposit of salivary calculus impinging upon and causing inflammation of the gum. (Black.)

Fig. 340.

Section of an upper molar, with deposit of calculus on its buccal surface, causing inflammation and absorption of the gum and lower border of the predental membrane and alveolar wall. (Black.)

root. The process is a gradually progressive one, but the rapidity of deposit and of alveolar recession varies widely. From beginning deposit

Fig. 341.



Sectional illustration of a heavy deposit of salivary calculus on a lower incisor, with partial destruction of the alveolus of the tooth. (Black.)

Fig. 342.

Sectional illustration of lower incisor with deposit of salivary calculus less heavy than that shown in Fig. 339. but with greater destruction of the alveolus. (Black.)

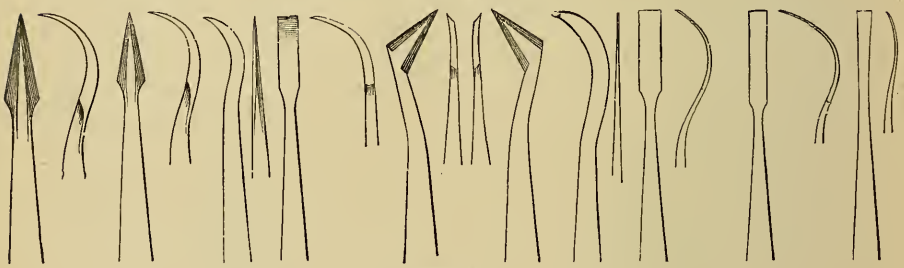
to almost complete loss of alveolar walls may occupy but a year or two; in other cases the atrophy of the alveolar walls is very slow. Infection of the pericementum may occur, when the loss of the tooth is much hastened.

The teeth become progressively looser until all bony connection is lost, being retained at the apex of the root by but few fibrous shreds. As soon as the alveolar loss is sufficient to cause marked loosening of the tooth, infection of the pericementum is common, and suppuration is grafted upon the results of mechanical irritation.

Prognosis.—The prognosis of this condition depends upon the extent of alveolar atrophy. If the loss of support be not so extensive as to cause marked loosening of the tooth or teeth, the teeth may be retained for an indefinite period, if they be so attached to neighboring teeth as to render them firm. If left unsupported, the pericementum is certain to degenerate, owing to the increased mobility. The alveolar atrophy will continue, and probably infection of the degenerated pericementum occur. Redeposit is almost certain unless all morbid conditions are removed and extraordinary precautions be taken as regards cleanliness.

Treatment.—The treatment may be divided under three heads: removal of deposits, correction of the effects of their presence, and prevention of their recurrence. The sole means of removing calculi should be instrumental. It is frequently recommended that mineral or some of the organic acids be used to soften the deposits to facilitate their

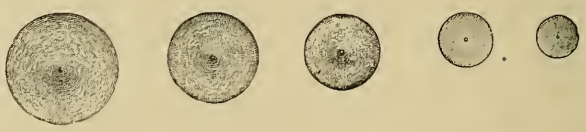
FIG. 343.



Scalers.

removal. Anyone, having seen a case in which a solution (5 per cent.) of sulfuric acid had been used for this purpose, needs no further warning against the application. Acid solutions will certainly soften the deposits, but at the same time inevitably cause a roughening of the enamel of the teeth by a solution of the calcium salts. To be sure, the

FIG. 344.



acid does affect the calculus more than it affects the enamel, but the roughened surfaces of the latter not only invite widespread deposits of

fermentable material, but render certain the more extensive accumulations of calculi in the future. The gross deposits may be removed by means of large sickle-shaped scales and curved chisels, nearly all used with a draw cut (Fig. 343). The instruments should have sharp edges and be introduced beneath the deposits, so that the gum is not unnecessarily wounded. The scaling should be continued until every surface which can be cleansed by these instruments is perfectly smooth. The case may then be dismissed for two days or longer; in the mean time an astringent

Fig. 345.

Fig. 346.

Fig. 347.

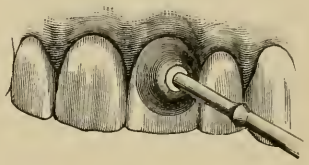


Fig. 348.

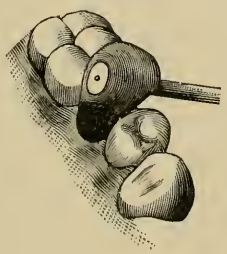


Fig. 349.

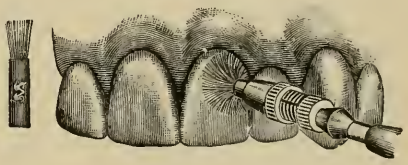
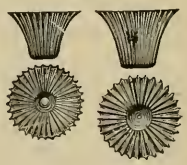


Fig. 350.

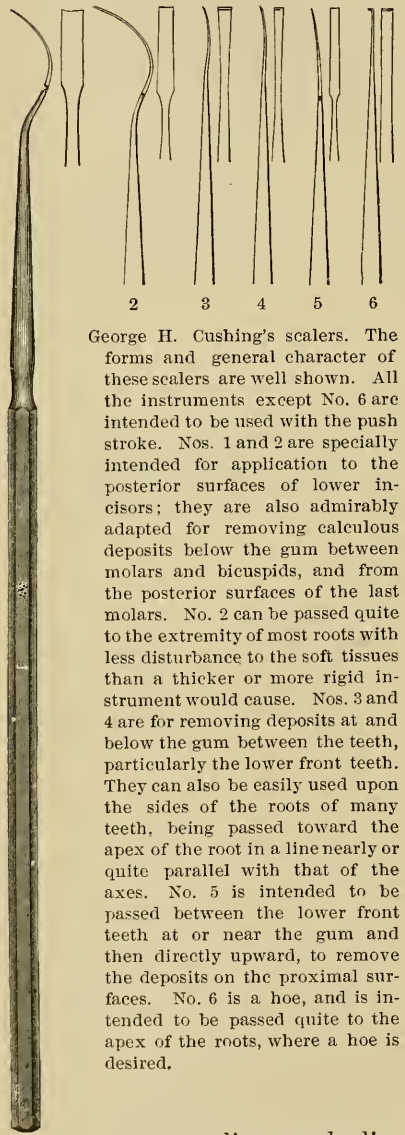
Fig. 351.



mouth-wash is to be freely used. The zinc-chlorid prescription (given on p. 447) answers admirably, or a mixture of equal parts of listerine and extract of hamamelis, diluted one-half with water, is serviceable. The passive congestion of the gums will be reduced and swelling lessened by these washes, permitting a better view of the surfaces of the teeth. More slender instruments of chisel-form which will pass into the spaces between the teeth and beneath the gum-margin, without wounding, are now required (Fig. 352). All of the calculi visible, and all that can be detected by their roughness, are thoroughly detached and scraped away by these instruments. The surfaces of the teeth are next cleansed with pumice made into a paste with glycerin. The paste is applied to the surfaces of the teeth, and the rubber cups and small brushes are used to cleanse the labial, buccal, and such lingual faces of the teeth as the brushes will reach (Figs. 345 to 351). The lingual surfaces of upper and lower incisors are cleansed with moose-hide wheels (Fig. 344) and wheel-brushes. The approximal surfaces of

the teeth are cleansed with linen tapes armed with the pumice-paste. The gums and teeth, as a final measure, are sprayed with 3 per cent. pyrozone to remove the pumice. It is advisable to repeat the polishing with precipitated chalk and the same carriers. The astringent mouth-wash is advised for a week's use; at the end of this time all evidences of gingivitis should have disappeared, unless small calculi still remain under some portion of the gum-margin, when their presence is denoted by the redness of the overlying gum.

Fig. 352.



George H. Cushing's scalers. The forms and general character of these scalers are well shown. All the instruments except No. 6 are intended to be used with the push stroke. Nos. 1 and 2 are specially intended for application to the posterior surfaces of lower incisors; they are also admirably adapted for removing calculous deposits below the gum between molars and bicuspids, and from the posterior surfaces of the last molars. No. 2 can be passed quite to the extremity of most roots with less disturbance to the soft tissues than a thicker or more rigid instrument would cause. Nos. 3 and 4 are for removing deposits at and below the gum between the teeth, particularly the lower front teeth. They can also be easily used upon the sides of the roots of many teeth, being passed toward the apex of the root in a line nearly or quite parallel with that of the axes. No. 5 is intended to be passed between the lower front teeth at or near the gum and then directly upward, to remove the deposits on the proximal surfaces. No. 6 is a hoe, and is intended to be passed quite to the apex of the roots, where a hoe is desired.

The smoother the surfaces of the teeth are made the longer will the redeposition of calculi be delayed. The operation described, so far as beneficial effects are concerned, is one of the most important in the practice of dentistry. No case should be dismissed before it is done; and no long series of operations should be begun before the cleansing. The patient should, in all cases, upon dismissal, be given explicit directions as to mastication and its importance, the wise regulation of the dietary, and the advisability and importance of using antiseptic mouth-washes.

Subgingival Deposits.

Definition.—By subgingival deposits are meant calculi which are first deposited in the annular depression between the gum-margin and a tooth. They are harder, smoother, and much darker than common salivary calculi, and collect in much smaller masses; they are not found upon the crowns of the teeth, their encroachment being in the direction of the cementum (Fig. 353).

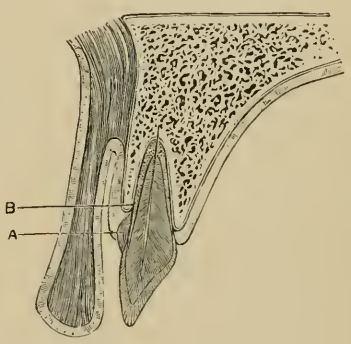
Composition.—So far as imperfect analyses have shown, these

deposits are composed of calcium phosphate combined with undetermined organic substances.

Occurrence.—The cavity in which they are found is open to the saliva. They are associated with marginal gingivitis, being found after a marginal catarrhal condition has been established, and before any direct evidence of degeneration of the pericementum is observed. So far as clinical observations can indicate, their formation is in consequence of the gingivitis; the primary causes of the deposit are, therefore, the causes of marginal gingivitis, both predisposing and exciting; these causes have already been discussed. Following the general rule of calculus-formation, the source of the deposits would be, then, the precipitation of the calcium salts of the saliva into the inspissated or coagulated altered secretion due to the catarrhal disturbance.

Effects.—The direct effects of the deposits are those of a persistent foreign body, a mechanical irritant in contact with vital tissue. The remote effect depends upon whether any of the causes recognized as predisposing to pericemental degeneration exist, such as anæmic, leukæmic, conditions; disuse, misuse, or overuse of the teeth, nephritic or arthritic disorders. Any and all predispositions in this direction, it will be recognized, are noted in that period of life known as the degenerative period, beginning in most persons after the fiftieth year; minor evidences of the advent of this period may be seen as early as thirty years of age, or even sooner. Predisposing causes, any of those named, existing, and active causes of gingivitis arising during this period of degeneration, thirty to fifty years: the calculi form, and incite degenerative changes in the pericementum, attended by a more or less constant set of symptoms, constituting a condition known as pyorrhœa alveolaris, better known in America as Rigg's disease. During the progress of pericemental destruction a fourth type of calculus makes its appearance, scattered as small islets over those portions of the tooth-root denuded of pericementum (Fig. 368).

FIG. 353.



A, subgingival calculus; B, receding pericementum.

Predispositions to pericemental degenerative changes may manifest themselves in limited atrophies. Unaccompanied by inflammatory symptoms, and by no evidences of local irritation, the normal-looking gums may recede from about the necks of the teeth, exposing a variable amount of the cementum; in molars the point of root-bifurcation is frequently exposed. The shrinkage of the gum may be confined to the

labial and buccal faces of the teeth, or the lingual aspects may also be involved. An atrophy of the alveolar margins has occurred and the point of pericemental attachment becomes progressively higher. This atrophy may affect one or more of the teeth. It is of frequent occurrence when the adjoining tooth or teeth have been lost, and the normal resorption of the alveolar walls at the points of extraction is not limited to these sites, but involves the alveolar wall of the tooth adjoining, and at but one aspect. The process is most marked in the lower second bicuspids after the molars have been lost. Secondary and more rapid degenerative changes may succeed the slowly advancing atrophy at any stage of its progress.

CHAPTER XXV.

PYORRHŒA ALVEOLARIS.

While the term pyorrhœa alveolaris implies but one symptom common to several distinct varieties of disease of the pericementum, that of a flow of pus from the alveolus, it is generally understood as a term descriptive of degenerative conditions which have some distinctive features; these are a progressive loosening of the teeth attended by a loss of the retentive structures, alveolar walls and pericementum, the loosening of the teeth being in a majority of cases attended by a flow of pus from the affected alveolus, and by deposits of calculi upon the denuded roots. The disease ceases spontaneously with the loss of the teeth; the resorption, loss, or atrophy of the alveolar wall being arrested at any period of the disease, if the affected tooth be extracted.

All of the varieties of this disorder, of which there are at least three clinical types, are associated with all of the predisposing and active causes of degenerations of the pericementum, and are most common from the age of thirty onward, although the disease may in exceptional cases appear during childhood, notably in rachitic patients.

Attempts to include all cases of tooth-loss, characterized by the features above named, into one class, based upon the pathogenesis of the disease, have thus far signally failed, and have done much to increase the confusion already associated with theories as to definition, causes, prognosis, and treatment of clinical cases.

Disposition has not been wanting to assign a specific form of infection as the causative element of this peculiar mode of tooth-loss, but thus far the attempts have been unsuccessful, although in all cases where pus-formation is found the pyogenic staphylococci and streptococci are undoubted attendants. Miller's experiments in this direction[1] failed to isolate any specific bacterium. He quotes largely from other experimenters, notably Galippe and Malassez, whose researches in the same field were all indeterminate.

Leaving aside questions of direct and remote causation, the cases may be clinically divided into three classes:

First, cases associated with and arising from a primary gingivitis, with the formation of hard, scaly, dark, annular calculi beneath the gum-margin.

[1] *Micro-organisms of the Human Mouth.*

Second, cases in which gingivitis may not be marked; the early deposits may be entirely absent; the necrosis of the pericementum advances in such a manner as to warrant the designation of Black—phagedenic pericementitis.

Third, cases in which the degeneration and necrosis of the pericementum and deposits of calculi occur upon some lateral aspect of a tooth-root, the gum-margin being apparently normal.

The differentiation between these several conditions is important, because while all three exhibit some features in common, they differ as to causes, clinical histories, prognosis, and mode of treatment. In the first class pericemental degeneration appears to be a secondary feature; in the second the distinguishing feature; and in the third degeneration and death of a circumscribed portion of the pericementum constitute the first evidence of the developed disease. They all agree in having the diseases of suboxidation as general predisposing causes of their occurrence; notable among which are the morbid conditions included under the head of the gouty diathesis; the last differs from the others in having a gouty condition as the probable exciting cause of the disease.

Pyorrhœa Alveolaris beginning as a Marginal Gingivitis.

Causes.—The causes of this condition are both predisposing and exciting. The predisposing causes are the causes, both general and local, of marginal gingivitis (which see), and the causes which give rise to debility of the pericementum—overuse, misuse, and disuse of the teeth.

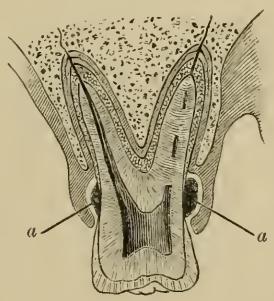
The exciting causes are, first, those of marginal gingivitis; secondly, deposits of calculi; thirdly, infection of the irritated tissues. At later stages of the disease other factors enter into the case and modify its progress; viz., looseness of the tooth and death of the pulp.

Symptoms and Clinical History.—The symptoms of the disease differ at the stages of its progress. As a rule, the disorder is most commonly seen after its first stages. The first stage is a marginal gingivitis; the gum-margin is swollen œdematous, and reddened; the extent of the swelling and discoloration varies with causes and individuals. In some cases the swelling may be pronounced and the gum purplish in color; in others swelling and discoloration may be but slight. At a later period an instrument passed beneath the gum-margin may detect the presence of a scaly collection of dark-green calculus, partially enclosing the neck of the tooth. Cases are seen where the formation of this calculus appears to have preceded any evident morbid condition of the gum. In many cases the hyperæmic condition of the gum-margin appears to lessen after a smooth calculus has formed. Apparently the calculus has formed in consequence of the union between the calcium

salts of the saliva and the inflammatory exudations, the vascular conditions afterward subsiding. Infection and swelling of the gum are, however, the usual condition. The swelling and discoloration persist; the calculus-formation encroaches further upon the pericementum, and the root becomes progressively denuded. As a rule, a recession of the line of the alveolar process is constant with the degree of root-denudation. At any period, and usually early, evidences of infection appear; pus may be pressed from beneath the gum-margin. Except in the later stages, or unless pus is confined, it is unusual to find the tooth particularly sore upon percussion. When about one-half or more of the root has been stripped of pericementum and deprived of alveolar support, looseness and extrusion of the tooth become marked. The teeth are nearly always looser than normal, even in the early stages.

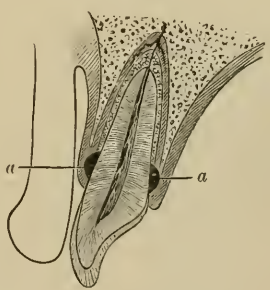
The advance of the disease now becomes more rapid; the undue mobility of the tooth excites an inflammatory reaction beyond the directly infected part, so that soreness and looseness are further increased. A scaler passed into the pocket formed between the gum and tooth will now usually detect marked roughness of the root. If the latter be scraped, small, hard, nodular deposits of calculi are detached with difficulty. After the looseness of the tooth becomes marked, the pulp of the tooth is usually killed by strangulation of its vascular supply. Infection of the dead pulp readily occurs, and septic apical pericementitis arises. The symptoms of the latter condition

FIG. 354.



Section of an upper molar with its alveolus, etc., showing deposit of serumal calculus under the gingival borders; *a*, *a*, serumal calculus. (Black.)

FIG. 355.



Section of an upper incisor, showing at *a*, *a*, a deposit of serumal calculus within the free margin of the gum. (Black.)

are modified, according to the facility with which the pus finds vent along the degenerating pericementum. The disease proceeds until the affected tooth or teeth are cast out, the alveolar walls and pericementum having entirely atrophied. The disease ceases with the loss of the affected teeth, leaving a flattened or absent alveolar ridge covered by a

mass of more or less spongy gum-tissue. The duration of the disease may be months or years.

A general subcatarrhal condition of the mouth usually attends the disease.

Pathology and Morbid Anatomy.—Figs. 354, 355, and 356 exhibit three distinct relationships of this form of calculus. Present data relative to the pathology and morbid anatomy of all three classes of pyorrhœa have been derived from clinical observations of teeth in the mouth and from an examination of teeth which have been lost at some period of the disease.

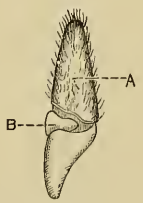
FIG. 356.

Section of an upper incisor, showing at *a* a deposit of serumal calculus and destruction of the lower border of the alveolar wall and peridental membrane, with a slight recession of the gum, exposing the calculus. (Black.)

THE TEETH.—In all of the varieties of pyorrhœa it is the general rule to find the teeth singularly free from dental caries. In a majority of cases their forms are of a type considered characteristic of the bilious and the nervous temperaments—broader at the occlusal surfaces and cutting-edges than at the necks of the teeth. Upon section all of the dental tissues are seen to be of a highly organized type; they offer unusual resistance to cutting-instruments. The enamel has a flint-like hardness, the dentin is much increased in translucency, and the pulp-chamber is much contracted—*i. e.*, the formative activity of the pulp has been carried to its extreme limit. The pulp-tissue is increased in density, and there is a notable increase in its fine fibres. One section exhibited an apparent loss of odontoblasts (atrophy) over a considerable surface of the pulp-periphery; others of these cells were reduced to comparative flatness. This, however, was a clearly gouty case.

The appearance of the root depends upon the stage of the disease when the tooth was lost. In the early stages a band of dark, smooth, scaly calculus surrounds a portion of the neck of the tooth, and embraces the cementum, slightly overlapping the enamel. Beyond the calculus is an area of denudation, the cementum free from pericementum; beyond this, the pericementum is intact, but its fibres are swollen, and its color deepened. The presence of a ring of bare cementum between the pericementum and the calculus was constant in all of the specimens examined. The inference is that after the primary deposit, necrosis of pericementum precedes

FIG. 357.



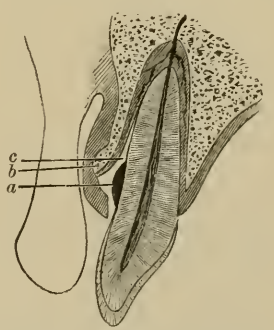
A, thickened pericementum; B, subgingival calculus.

subsequent deposits. Teeth extracted at later periods exhibit usually two varieties of calculus: first, that which formed beneath the gum-margin; second, beyond, upon the denuded cementum, are small islets, bead-like deposits of rough, hard, dark calculi, which appear also in other dental conditions attended by continued pus-formation. They appear identical with the deposits which are found upon the apices of roots in long-continued chronic septic apical pericementitis (alveolo-dental abscess), and on the sides of roots in cases of the same disease discharging at the gum-margin, in cases of phagedenic pericementitis, and as secondary deposits in gouty pyorrhœa. Their association with chronic pus-formation appears clear, their presence being evidence of continued suppuration. They are to be regarded as resultant from, not causative of the pericemental degeneration. They are more often present in cases of delayed than in rapid tooth-loss. Teeth extracted at the latest stages of the disease show that the apical pericementum still maintains attachment, and is much swollen. Examining the tooth or teeth before they are extracted, a pocket is found to exist beyond the calculus, and its direction and situation depend upon the position of the calculus. If this be, as it frequently is, upon the palatal surface of the root, the formation of a pocket at the labial aspect may not be marked until the posterior pocket is much deepened.

In most of the cases of the first class of pyorrhœa a probe fails to discover uncovered alveolar bone, although it may do so; a matter of some importance, as it indicates in some measure the mode of alveolar degeneration. If the bone be covered, its resorption is more of the nature of an atrophy; if uncovered, it is probably lost in part through peripheral molecular necrosis. The conditions point to a progressive recession of the pericementum, with the loss of alveolar wall, as an atrophy, a secondary process.

It is to be remembered that all evidences of alveolar disease cease promptly with the loss of the affected teeth.

Fig. 358.



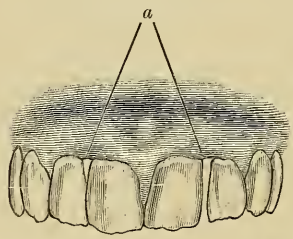
Section of an upper incisor, showing destruction of the peridental membrane and eversion of the alveolar wall, with thickening of its border: *a*, serumal calculus; *b*, thickened border of the alveolar wall; *c*, pus-cavity. (Black.)

A condition sometimes met with is recorded by Black, in which a dual periosteal disease is in evidence (Fig. 358). The irritation caused by the presence of a subgingival deposit has resulted in an atrophy of the immediately adjacent alveolar process; at the labial aspect of the alveolar edge the pericementum has been irritated to a constructive stage and a new deposit of bone has occurred, resulting in

a distinct thickening of the alveolar margin. This condition illustrates well the effects of grades of irritation; how one grade is productive of increased functional activity, another of degeneration and atrophy. It is rare for this variety of pericemental destruction to proceed to any considerable depth without abundant manifestations of pyogenic infection. The infection, however, exhibits no disposition to invade the outer maxillary periosteum; it follows the direction of the pericementum, and ceases promptly as soon as the teeth are extracted, no matter at what period.

Diagnosis.—Usually but little difficulty attends the diagnosis of this varety of pyorrhœa. As a rule, several teeth are affected; those in in which the characteristics of the disease are most marked are the upper incisors. If the disease is fully established, it will be confounded with no other condition: the tumid gum, the presence of easily discoverable calculi in a subgingival pocket, and the oozing of pus, all furnish a clinical picture not associated with any other dental disorder. The occurrence of these pericemental degenerations can be foreshadowed at times. In any of the three forms, evidences of marginal atrophy of the alveolar walls may be noted before any deposits of calculi can be detected, or before there are any indications of the initial gingivitis; the septum of gum between the teeth recedes, obliterating the normal gum-festoon (Fig. 359).

Fig. 359.



Absorption of the septum of bone and recession of the gum between the central and lateral incisors, caused by deposits of serumal calculus under the gingivæ. (Black.)

The existence of a persistent gingivitis, with an exaggeration of the space between tooth and gum-margin, means a future pyorrhœa.

Differential Diagnosis.—In the later stage care will be necessary to determine which variety of pyorrhœa is present. In the first variety the bottoms of the pockets are usually found, not far beyond evident deposits of calculi. Indications of the existence of an annular alveolar wall are also observed. Gingivitis keeps pace with the loss of pericementum. In phagedenic pericementitis the deposits may only be recognized with difficulty; the degeneration proceeds in a direct line toward the apex of the root; the pericementum may still be attached along the other portions of the root; the gingivitis may be very slight; the gum-edge although receded, may retain its normal festoon. In gouty pyorrhœa (proper), deposits about the neck of the tooth are unusual, and, except in later stages, the gingival attachment appears almost normal. A slender instrument is passed with some

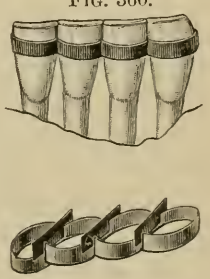
difficulty beyond the gum-margin; its introduction may, again, be entirely resisted. Pressure upon the tooth shows it to be loose, and pressure over the gum near the apex may show a boggy softness due to an outlined loss of alveolar wall. The family and personal history is of great importance.

In gouty pyorrhœa, and in phagedenic pericementitis but few teeth are attacked at once, as a rule; frequently but one tooth is affected; in the first variety several teeth are affected simultaneously.

Prognosis.—Of all the varieties of pyorrhœa, the first is that having the most favorable prognosis. If properly treated medicinally and mechanically, proper directions given the patient, and frequent supervision exercised, the disease may be arrested in any but its latest stages. If predispositions to pericemental degeneration exist, they will modify the prognosis in the degree of the difficulty of eliminating them. The assertion is made by many practitioners that in their hands pyorrhœa alveolaris is an entirely curable disease. These assertions refer, no doubt, to the first class, for, as will be seen, the prognosis of the second and third classes of pyorrhœa may be independent of therapeusis.

Treatment.—From the point of view of therapeutics, several conditions exist in the morbid phenomena described. First, infection; second, the presence of foreign bodies; third, teeth in any degree of partial luxation; fourth, mal-occlusion; fifth, vascular disturbance of the gum and pericementum; sixth, probably an underlying constitutional predisposing cause. Treatment is directed, first, to a removal of all sources of irritation. The sources of offence are to be removed in the order of their danger to the teeth. If the disease have progressed far, the imminent danger is mechanical dislodgement of the loosened teeth; the affected tooth is to be lashed to its firmer neighbors, so that it is immovably held. A careful examination is next made of the occlusal relations; in the majority of cases the occlusion will be found excessive. The teeth and in some cases their antagonists are ground away by means of corundum wheels until they are slightly short of occlusion. The exposed portions of the crowns and roots are scaled free from all deposits. As it will be necessary to hold the affected teeth immovably during the period of healing, the nature of the splint required is determined upon. If but two or three anterior teeth are to be held, rings made of thin platinum plate form effective splints (Fig. 360). The teeth to be splinted are ligated firmly together at their necks, and a strip of platinum plate, No. 34, is annealed and moulded against the lingual faces of the teeth; it is passed between the teeth to be splinted and

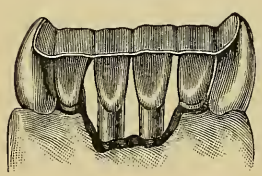
Fig. 360.



those posterior, and its free ends are overlapped; a scratch is made to indicate the overlap, and the piece is detached and soldered. Returned to the teeth, the thinnest separating saw is passed between the teeth, grooving the splint deeply upon both sides; in these grooves straight strips of platinum plate are placed. If the piece can be lifted from the teeth without force, it is so displaced; if not, a plaster impression is taken, a cast of investing material made, and the strips soldered in their grooves. The investment and splint are thrown hot into sulfuric acid, which cleanses the splint and removes the investment without force. The splint is washed in running water, the teeth are wiped off with chloroform, and the splint cemented in place with zinc phosphate.

Where several teeth are to be held a swaged plate is usually the most effective splint. After the occlusion has been overcorrected, a plate to cover the loose and the immediately adjoining teeth is swaged and cemented to the teeth (Fig. 361).

Fig. 361.



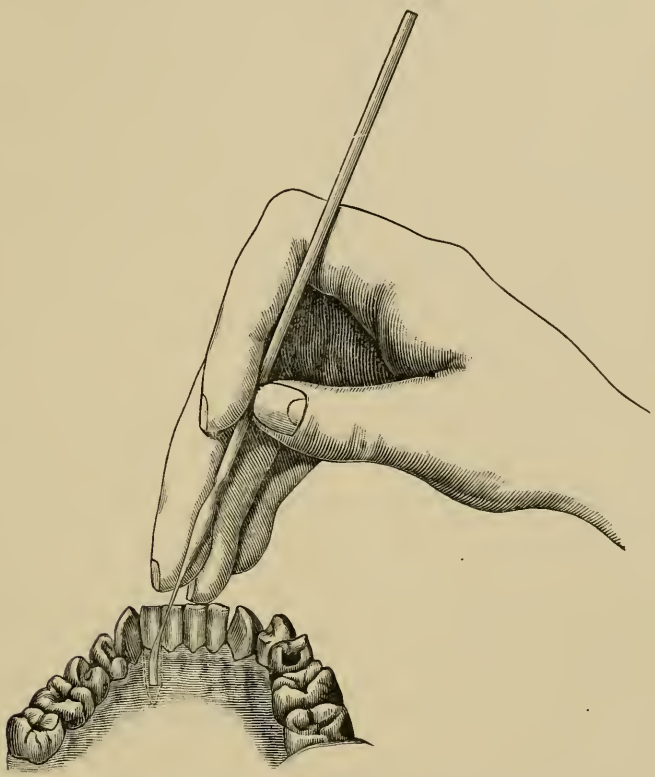
The operator's ingenuity will show him which of the great number of retaining appliances devised will serve best in a particular case. Many of the retainers used in orthodontia serve admirably as splints. The splint in position, surgical rest is assured. The agent second in importance, as threatening the pericementum, is the infection. Antiseptics are to be freely used during all operations and frequently by the patient after, and between operations. The pockets are first freely syringed or sprayed with hydrogen dioxid until effervescence ceases; wisps of cotton dipped in 25 per cent. pyrozone are next placed in the pockets (Ottolengui); a white eschar forms at once, and any pus or decomposing organic matter remaining is destroyed. The next threatening element is the calculus; so long as deposits of calculi remain, irritation will continue. Beginning with the tooth most threatened, delicate scalers (Fig. 362) are passed beneath the gum, and used with a push cut to chisel away all deposits. The instruments must be delicate in order to detect the granule-like deposits. The cutting is to be firm and decided, but slipping of the instrument must be guarded against by resting the fingers upon the tips of the teeth (Fig. 363). The scraping is

Fig. 362.

Showing the application of a thin, flat instrument to the labial and approximal surfaces of an upper bicuspid (pushing motion).

alternated by syringing with hydrogen dioxid to cleanse the pockets. If much swelling of the gum be present, tampons of cotton saturated with 10 per cent. solution of trichloracetic acid packed in the pockets

FIG. 363.



Showing the manner of holding an instrument for detaching calcareous deposits when using the pushing motion. The third finger rests on the edges of the teeth, allowing freedom of the hand to make rapid and effectual movements in dislodging the calculi.

for a few minutes will check oozing and permit a better view of the pockets.[1] The scaling of one tooth is to be completed before passing to a second tooth. This, however, does not apply to the first visit after splinting the teeth; as it is frequently advisable to then remove the coarse subgingival deposits from all of the teeth, cleanse the pockets with 25 per cent. pyrozone, and prescribe an astringent antiseptic mouth-wash to be used several times daily for two or three days:

℞. Zinc chlorid,	gr. x;
Aq. menth. pip.,	℥j.—M.

The tumefaction of the gum will be much reduced and pus-formation

[1] Kirk.

checked by this means. At the second visit the scaling should be begun and continued upon a tooth until it appears entirely free from roughness before passing to a second, and so on. Partial scaling, repeated at intervals, interferes with or prevents the regenerative process. After each scaling the pockets are syringed forcibly with hydrogen dioxid. The use of acids, particularly two of the organic acids—trichloracetic (Kirk) and lactic (Younger)—is advised; they have been found serviceable as an application after scaling and syringing. They act as solvents upon the minute calculi which escape the scaler, and as caustics, stimulating healthy action in the ulcerous pericementum. Unless pus-formation or congestion persist, the scaled teeth are to remain undisturbed. After the operation, the use, several times a day, of the astringent and antiseptic mouth-wash given above is advised.

Truman advises the use of hydronaphtol in an astringent vehicle as an effective germicide for use by a patient:

℞. Hydronaphtol, gr. x;
Glycerol, ℥j;
Alcohol, ℥j;
Aq. destill., ℥ij.—M. (Peirce.)
S. Use as wash several times a day.

In case of recurring or persistent pus-formation the pocket should be again explored, cleansed and dressed as described.

The splints are to remain in position until the gum appears to grasp the teeth firmly, is of normal color, and pockets have closed. If more than half of the root has been denuded, permanent splints are advisable. Regeneration of alveolar process does not occur; the gum gradually shrinks to its normal relation with the process in its atrophied condition.

Constant care upon the part of the patient, both as to general and oral health, and occasional inspection by the operator are necessary for even reasonable assurance against a recurrence of the disease. Recurrence is more or less probable, no matter what precautions are taken. Even when pus- and calculus-formation are not re-established, atrophy of the alveolar process is common. The advent of these diseases expresses a degenerative impulse against which the best of therapeusis frequently fails, almost entirely.

Phagedenic Pericementitis.

The term phagedenic pericementitis was suggested by Black to designate a condition whose most prominent feature is a progressive death of pericementum, beginning at its marginal attachment, not caused by me-

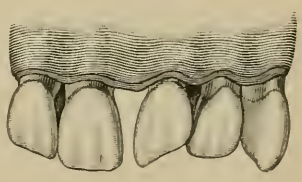
chanical injury, chemical agencies, specific virus, or the selective action of drugs; and whose usual, although not constant, accompaniments are pus-formation and deposits of calculi. Its progress is phagedenic, and ceases with the loss of the tooth. From the pus-flow this disease has been included under the generic head of pyorrhœa alveolaris, although it is clearly distinguished from the disease already described as due primarily to subgingival deposits.

Definition.—In light of present data phagedenic pericementitis may be defined as a condition which comprises degeneration, atrophy, and molecular necrosis of the alveolar process and a molecular loss of pericementum, accompanied in its developed state by pyogenic infection and usually by deposits of nodular calculi. The pericementum and alveolar process covering a root may be entirely destroyed without any distinct evidence of inflammation, suppuration, or calculus-formation.

Causes.—The causes of this condition appear to be divisible into predisposing and exciting. The predisposing causes are those named as productive of pericemental debility and degeneration. Among the general predisposing causes are heredity, particularly as to arthritic diseases, and the diseases of suboxidation and faulty elimination. Among the local predisposing causes are overuse, disuse, and misuse of the teeth. Overuse and misuse often act as direct exciting causes. The primary gingivitis noted as inviting the first variety of pyorrhœa alveolaris, appears to play a subordinate part in the causation of phagedenic pericementitis, although it does usher in the disease in some cases, when a deposit of subgingival calculus appears to act as the exciting agent of the degeneration.

Symptoms.—It has been maintained that this disorder is essentially infective, but thus far all attempts to discover organisms which are pathognomonic have failed. It is more than probable, however, that future studies will discover in bacteria an acute etiological factor in the disease. The symptoms most characteristic and distinctive of this disease are best noted upon an upper central incisor, and others upon the palatal root of an upper molar. At a period antedating any evidences of pericemental affection a tooth, frequently an upper incisor, shifts its position, moving outward, or rotating and separating from one of its neighbors (Fig. 364). There is no peculiarity of the occlusion which will explain this shifting. At a later period—it may not be for many months—the shifted tooth is seen to be looser than its neighbors. This loosening is hastened if, in shifting, a condition of mal-occlusion is established. Before the tooth loosens, evidences of

Fig. 364.



atrophy of the alveolar margins may be seen in a recession of the gum-festoon between the teeth. As soon as looseness is marked, evidence of pericemental loss may be detected by passing an instrument beneath the gum-margin, which is seen to be detached at some point, commonly between the teeth where the gum-recession was first noted, or at the

Fig. 365.

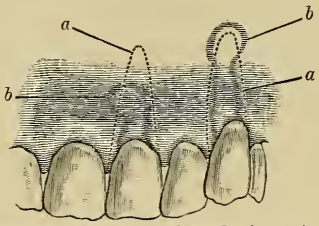


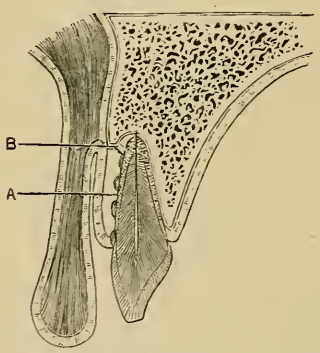
Illustration of a case of phagedenic pericementitis: *a*, *a*, dotted lines representing the outlines of the roots of the teeth; *b*, *b*, irregular lines representing the extent of the destruction of the peridental membrane and walls of the alveolus. It will be noted that the gums appear nearly perfect. (Black.)

Fig. 366.

The same case shown in Fig. 365, denuded of the soft tissues to show more plainly the loss of the walls of the alveolus. This drawing was made after raising a semicircular flap of the soft tissues over each root for the purpose of thorough exploration. (Black.)

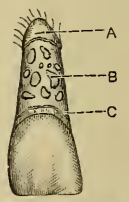
labial aspect of the root (Figs. 365 and 366). While the gum-margin usually presents signs of irritation, it may be apparently unaffected. If gingivitis be, or have been, present, subgingival calculi will probably exist. Exploration of the pockets will show them to be the consequence of death of pericementum, which has followed the length of this structure along the side affected; the remainder of the pericementum and alveolar process may be intact. The root may exhibit no roughness what-

Fig. 367.



A, calculi of pyogenesis; B, ulcerous pericementum.

Fig. 368.



A, thickened pericementum; C, subgingival calculus; B, calculi of pyogenesis.

ever. A careful exploration usually shows a portion of the atrophying alveolar wall over the necrotic area to be denuded; the pericemental death has proceeded more rapidly than alveolar atrophy. The pockets increase in size, and the alveolar process disappears until the teeth can be extracted with the fingers. It is unusual for the pockets to attain any considerable depth before evidences of pyogenic infection occur; so that

pus may usually be pressed from the pockets. In cases where pus-discharge is present, the small nodular calculi may be detected upon the roots.

When pericemental destruction has involved the apical pericementum death of the pulp occurs, and infection of the necrosed pulp results; abscess forms and pus discharges *via* the pyorrhœa pocket.

When the disease attacks but one root of a molar, destruction of the pericementum around that root, death of half of the pulp, and abscess-formation may result, and the other roots be unaffected; a portion of the pulp of the tooth may retain its vitality for some time, notwithstanding the apical abscess upon one root. In these cases pus-discharge from about the root of a tooth may be continued by the infected dead pulp, after all pericementum and alveolar process are gone from about the root.

While the disease is usually first noted about a single tooth; it is rare that a lengthened period elapses before it makes its appearance about other teeth; usually an adjoining tooth, or, it may be, on a distant one. This disease may make its appearance in the mouths of patients who take extraordinary care of the teeth, in mouths where the teeth are apparently entirely free from deposits, where the gum appears normal, and where the teeth are free from caries. It is of more frequent occurrence in dentures comparatively free from caries than in those where caries prevails or has prevailed.

Morbid Anatomy and Pathology.—Teeth lost through this disease may exhibit no abnormal appearances at all, except the entire absence

FIG. 369.

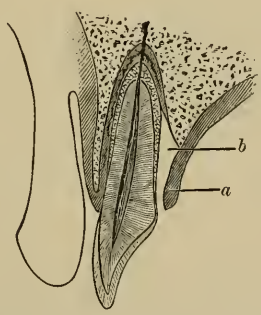


FIG. 370.

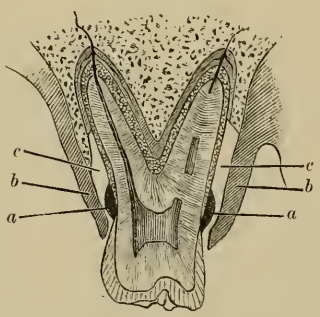


Fig. 369.—Section of upper incisor, showing destruction of its peridental membrane and alveolus by phagedenic pericementitis: *a*, gum-tissues covering pus-cavity (*b*) formed by the destruction of the peridental membrane and alveolar wall. (Black.)

Fig. 370.—Section of an upper molar, showing destruction of its membrane and alveolar wall by phagedenic pericementitis: *a*, deposit of serumal calculus; *b*, *b*, gum covering pus-cavity (*c*, *c*) formed by the destruction of the peridental membrane and alveolar wall. (Black.)

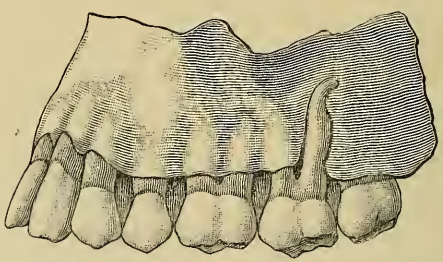
of even shreds of pericementum. They may be entirely free from all deposits. The teeth are nearly always of the type assigned to the bil-

ious and sanguine temperaments, and are singularly free from dental caries. The enamel of the teeth may be abraded in some degree, in which case the areas are seen to have a glossy polish. Upon section enamel and dentin are seen to be of the highest type of organization.

The pulp-chamber is much contracted. Data are wanting relative to the condition of the pulp in these cases; but it is inferred from the condition of the pulp-chamber, and the age at which the disease makes its appearance, that atrophic changes are probably present.

An examination of alveoli and pockets will show two types of the disease: those affected by the subgingival deposits and those without (Figs. 369 and 370). The root is denuded of pericementum to an unusual depth, and the edge of the alveolar process is found to be bare. A pocket may extend to the very apex of the root upon one side of a root, and the attachment upon the opposite side remain normal for a long period (Fig. 371). The alveolar septum between two adjoining teeth may be destroyed together with the corresponding portions of pericementum, and the remainder of pericementum upon both teeth remain intact for a long period. In cases without deposits an absence of gingivitis is frequently noted; even more, an atrophy of the gum-tissue occurs which bares to some extent the denuded root. Where subgingival deposits are present, gingivitis is the rule. The effects of the occurrence of constructive pericementitis upon the outer alveolar margin may be noted in some of the long-continued cases.

FIG. 371.

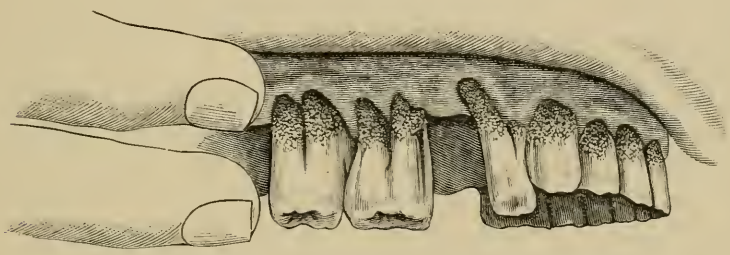


Showing loss of pericementum and alveolar process over one face of one root of a molar tooth from phagedenic pericementitis. Several of the other teeth were the seat of the first variety of pyorrhœa.

What was said of the first variety of pyorrhœa, relative to the location of the disease, applies with equal force to phagedenic pericementitis. It is a disease of the pericementum, and alveolar necrosis is a secondary feature, which ceases as soon as the teeth are lost by extraction or by the progress of pericemental necrosis. There is an atrophic variety of the disease which exhibits peculiar features. Marginal alveolar atrophy is seen to occur about the roots of several teeth. There is no vascular disturbance in the gum-tissue, so that the gum-line

recedes with the alveolar wall. The cementum of the root becomes exposed almost always at the labial or buccal aspects. Later, death of an annular portion of pericementum and infection by pyogenic organisms occur; immediately beneath the gum-margin small, hard, nodular calculi are formed. Instead of deep pockets, the pericemental loss is attended by a constant recession of the gum-line until the roots of the teeth become exposed for a great part of their length; the recession of the gums exposes the calculi which have formed (Fig. 372).

Fig. 372.



The alveoli irreparably destroyed by calcic inflammation. (Black.)

This process is far from uncommon in the mouths of persons who possess fine dentures, but who neglect the care of their mouths.

Diagnosis.—The differential diagnosis of phagedenic pericementitis from the first and third varieties of pyorrhœa has been given in connection with the first variety. Its direct diagnosis consists in discovering the peculiar pockets, denuded roots, and alveolar edge. Its occurrence can almost be certainly foretold in the mouths of patients who have fine dentures of the type described, who have particularly an arthritic history, and who, about the age of thirty or later, note the change of position of one or more teeth without evident cause. It will be recalled that the pockets in phagedenic pericementitis have a depth out of all proportion to their lateral extent.

As pointed out by Black, the condition may be confounded with apical abscess opening along the side of a root. If the latter be acute, the acute symptoms of alveolar abscess, absent in phagedenic pericementitis, point to a diagnosis. If chronic, there may be noted evidences of pulp-death, absent response to thermal stimuli, and the presence of large fillings. An examination should be made of the other teeth, for it is unusual that phagedenic pericementitis will progress to the end of the root of a single tooth before other teeth are involved. Doubt will be dispelled by opening the affected tooth, whether carious or non-carious. If the pulp be alive, it is phagedenic pericementitis; if dead, it may be that or alveolar abscess. If the latter, a cure is accomplished by treatment as described in Chapter XXII.

Prognosis.—The prognosis of this disease, so far as the teeth affected are concerned, is in general decidedly unfavorable. While it may be temporarily arrested in its earlier stages, its recurrence and ultimate loss of the affected teeth are the rule. It may attack but few teeth of a denture and progress until they are lost, the other teeth remaining unaffected. The common history, however, is that when the disease makes its appearance the denture is ultimately lost through it, although the period of loss may cover many years. Several years may elapse between the loss of one tooth and the affection of the second. Upper incisors and molars appear to suffer more frequently from the disease than any of the other teeth.

Treatment.—Treatment of the case, based upon the relief of discoverable morbid conditions, will serve to stay the process. The conditions demanding correction are, faulty occlusion, undue mobility, the presence of necrotic tissue, usually an infection and foreign deposits. These latter are to be regarded as pathogenic in that they prevent a return to health of the gum-tissue overlying them.

The treatment is both prophylactic and remedial. Patients having an arthritic history and the type of denture named should be warned of the dangers of establishing a pericemental debility through relative disuse of the teeth, and of permitting morbid gingival conditions to arise from neglect of oral hygiene. The relations of food-habit and general disease, notably those of suboxidation, to dental disease are to be pointed out.

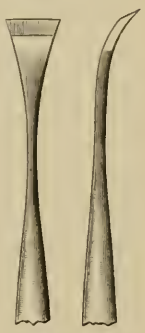
In the early stages of pyorrhœa, that of tooth-shifting, it has been asserted[1] that, if the tooth be opened, the pulp destroyed, and canal filled, the impending degeneration and necrosis of the pericementum will be averted. There is good clinical evidence, both of the writers quoted and others, to substantiate the assertions. The probable explanation is that the diversion of the apical blood-supply entirely into the pericementum protects this structure against threatened degeneration. There is reason to believe, although histological data in this connection are wanting, that changes in the bloodvessels and nutrition of the pericementum are antecedents to the degenerative and necrotic changes described. When the pockets have formed and alveolar atrophy is marked the conditions resemble in some particulars, but are by no means identical with, those noted in connection with the first variety of pyorrhœa.

The treatment, as regards splinting of the teeth and sterilization of the pockets, is the same as in the first class. Black emphasizes two points of much importance in the next stage of treatment—*i. e.*, the removal of deposits—first, that the gum-margin must not be unnec-

[1] M. L. Rhein; D. D. Smith.

essarily injured; secondly, that vigorous scaling of the roots may be done without special regard to avoid cutting the tissues lining the pocket, instead of avoiding such injury, as in the first class of pyorrhœa. The pockets are freely syringed with hydrogen dioxid, or with a 1 : 500 solution of mercuric chlorid in hydrogen dioxid. The alveolar edges are to be freely scraped with the scaling instruments, which should have slender stems and comparatively broad cutting-blades (Fig. 373). The use of cauterants, such as trichloracetic and lactic acids, is more important than in the former type of disease. The same astringent antiseptic washes are to be prescribed. After removing all foreign material, including dead matter and sterilizing, correcting occlusion, and securing immobility, the astringent antiseptic wash is expected to draw the tissues tightly about the teeth and to prevent infection, so that a regenerative process can be established in the vital tissues of the former disease-pocket.

FIG. 373.



Scalers (three times natural size).

In case the pockets are so deep or have such form that the alveolar margins cannot be well trimmed without overstretching or injuring the gingival edges, Black advises that gum-flaps be raised, exposing the alveolar margins (Fig. 374). A semicircular incision is made and turned back, and bleeding checked. By means of sharp chisels the alveolar borders are freely scraped, the pockets are flushed with hydrogen dioxid, and the flap secured by a couple of stitches. The same writer advises in cases where eversion of the alveolar margin has occurred, that the process be exposed by cuts and broken down by three cuts made with a sharp chisel and mallet; the loosened segment of bone to be pressed firmly against the root. It is desired next that the entire pocket will fill with granulation-tissue, and organization of the granulations take place, furnishing re-attachment. That this occurs in some cases is undoubted. Black believes that a reproduction of alveolar margins also occurs in some cases. The hope of good results lies in keeping the parts aseptic after all foreign deposits and dead material have been removed.

FIG. 374.

Illustration of the position and form of incision through the gum for exposing the root of the tooth and injured alveolar process; *a*, incision. (Black.)

A great number of agents have been advised as medicinal applications to the disease-pockets. They are all antiseptic and most of them cauterants. These are to be used after the primary cleansing. During

the period of granulation no solutions stronger than stimulants should be used. A 20 per cent. solution of zinc iodid (Harlan), the hydronaphthol solutions (Truman), and the zinc-chlorid wash primarily given may all be taken as representative medicines. The indiscriminate use of strong solutions of antiseptics retards the granulating process. The silver salts, lactate and citrate, 3 per cent. solutions of silver nitrate, and powdered nosophen should be mentioned as useful agents in this connection, particularly the silver salts.

The records of experiments with sponge-grafts are not encouraging, there being too great difficulty in maintaining them sterile.

CHAPTER XXVI.

DISEASES OF THE PERICEMENTUM BEGINNING UPON A LATERAL ASPECT OF THE TEETH.

Gouty Pericementitis.

From its common occurrence in persons who are the victims of the gouty or arthritic diathesis, a third variety of pyorrhœa alveolaris has been designated as gouty pericementitis.

Definition.—Gouty pericementitis may be defined as a condition in which degeneration and necrosis of the pericementum begin in some portion of that structure between the apex of a root and the gum-margin, usually attended by a deposit of calculus in the disease-area, which exhibits a combined reaction of urates and of calcium phosphate. As noted in connection with the first and second varieties of pyorrhœa, many general diseases included in the family of diseases called gouty, act as predisposing factors in their causation. In the third variety, not only does a gouty condition act as a predisposant, but it appears to furnish also the exciting cause of the disease.

The pathology and pathogenesis of the disease will be more evident after a survey of its clinical history and symptoms, and a review of the mode of action of the gouty poison. For many years gout and the gouty diathesis have been recognized as causative of a number of local, including among them, several dental diseases, periodical dental neuralgias, pericementitis, and phagedenic destruction of the pericementum. In 1886 W. J. Reese[1] pointed out clearly the association of the gouty condition with what he termed "phagedena pericementi." In 1891 J. S. Marshall demonstrated the analogy of certain pericemental degenerations with a gouty condition. Impetus was given the study of this association when, in 1892, C. N. Peirce demonstrated that deposits found upon the lateral aspect of the pericementum gave a murexid reaction, proving the presence in them of urates, the salts found in gouty concretions in other parts of the body. He also pointed out that the deposits occurred without primary destruction of the marginal pericementum, so that the primary disease-focus is found upon some portion of pericementum lying between the root-apex and gingival attachment.

Symptoms and Clinical History.—The symptoms of gouty dental diseases depend upon the stage at which they are seen. In the earliest stages vague functional disturbances appear before there is any evidence

[1] *Dental Cosmos.*

whatever of structural changes. Like other gouty affections, dental gout rarely makes its appearance before thirty years of age, and most frequently between forty and fifty-five years.

Beginning with the earliest visible evidence of disorder, dental gout appears to exhibit itself about as follows: in the mouths of persons presenting a clear family history of gout, or of rheumatoid arthritis in the female line, who have been the victims of gout, evident or obscure, more often the latter, or, again, suffer from the condition called "lithæmia" (professional man's gout), it is commonly noted that the teeth are singularly exempt from dental caries. This is a general, although not universal truth. The teeth are frequently of highly organized type, and of the variety said to be indicative of the bilious or nervous temperament. Frequently they are the seat of mechanical abrasion; their occlusal faces may be worn to any extent, the character of the wearing depending upon the nature of the occlusion; it is less marked in teeth having long cusps than in those having an originally short overbite. The long cusps show the spots of enamel-wear as areas having a glossy polish. Erosion (which see) is of frequent occurrence in such dentures, particularly among females. The dentures are of the class in which phagedenic pericementitis is apt to make its appearance.

In the mouth and about the jaws of such an individual, after the age of thirty, neuralgic pains may be of nightly occurrence; these pains are vaguely referred to the teeth. An examination of the teeth exhibits no direct reason for their occurrence; there may be no dentin-exposure and no tenderness upon percussing the teeth. It will be noted, though, that the response of the teeth to thermal changes is decidedly increased: a general heightened sensitivity of the dental pulps exists. These pains and the pulp-hypersensitivity disappear after a time. In some cases this may be spontaneous; in others it will be found that the patients have been receiving general medical treatment, and the maxillary pains disappear with the cure of the disorders treated by the general practitioner. At later periods occasional attacks of general pericemental tenderness may occur, which make their appearance and disappear as did the maxillary neuralgia. In both cases there is a return to apparently complete normality. These symptoms are also of frequent occurrence in the mouths of patients who suffer from dental erosion and abrasion. The exposed dentin in these cases furnishes a tangible cause for the reflex pains about the jaws.

If the teeth be examined after this period, the comparative exemption from caries, and usually from calculous deposits, is noted, together with the firm fixation of the teeth in dense alveolar process. However, a slight recession of the gum-line may be noted, as though a very limited marginal alveolar resorption had occurred. Later, periods of

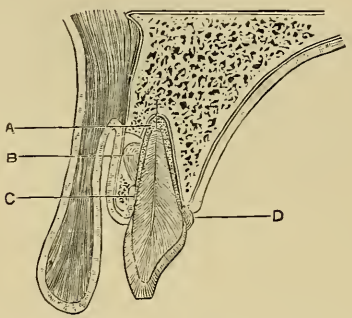
pericemental soreness may be more frequent, and one or several teeth during this period are loosened from their former firm implantation. The soreness may disappear, but the slight looseness of the tooth may remain.

It is at later periods and in some single tooth, that degenerative and necrotic changes become unmistakable. Some one tooth of a denture becomes sore and tender upon percussion; the gum overlying the apical half of the root exhibits the evidences of an underlying inflammation. The tooth is loosened, but the attachment of the gum-margin remains unbroken; indeed, unless the inflammation be marked there may be no evidence of marginal gingivitis. The symptoms closely resemble those of acute septic apical pericementitis, although less in degree. The inflammation may subside and leave the tooth permanently loose. In other cases a circumscribed swelling appears, indistinguishable from that of acute apical abscess; the surrounding inflammation is, however, less. If an incision be made into this swelling, a glairy, mucus-like discharge may vent; in other cases a flow of pus is observed. In the latter case pyogenic infection is certainly present; in the other, infection is uncertain. Many of these cases have been diagnosed as septic apical pericementitis due to dead pulp: the tooth being probably non-carious, the death of the pulp is assigned to one of the conditions described under diseases of the pulp. Upon drilling into the tooth dentinal sensitivity may appear to be absent, and the instrument may be plunged into a vital pulp—*i. e.*, the inflammation and suppuration have no connection with a dead pulp. If the incision into the abscess-cavity be enlarged and bleeding checked, it will be seen that a portion of the alveolar wall has disappeared, exposing the side of the root of the tooth, which is found to be denuded of a portion of its pericementum, and on the root rough bodies are noted, which if scraped away, are seen to be calculi.

In some cases the symptoms of pericementitis persist without the formation of a circumscribed swelling over the root, and later a discharge of glairy, mucus-like material or of pus exudes from beneath the gum-margin. The same errors of diagnosis, and discovery of live pulp noted above, are frequently made. In other cases the pericemental destruction may be so extensive and accompanied by a disappearance of such an amount of alveolar process that the teeth are very much loosened. If the teeth be non-carious, as they usually are, and the looseness have not been marked, an examination of the interior of the tooth shows uniformly the presence of vital pulp. Teeth have been extracted during this period, one of which exhibited these significant features: the apical pericementum was intact, as was also that portion toward the gingival margin; between the two was an area of denudation, in which, loosely attached to the root, was a rough, irregular calculus (Fig. 376).

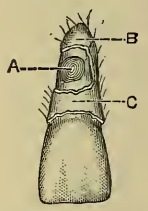
Calculi scraped from the roots of such teeth exhibit in a varying degree a response to the murexid test, the test for urates. The reaction may be

Fig. 375.



A and C, vital pericementum; B, gouty calculus; D, a subgingival calculus.

Fig. 376.



A, calculus in area of necrosis; B and C, vital pericementum.

very faint in some cases, being overshadowed by the calcium phosphate, which makes up the bulk of these masses; in others it is pronounced—*i. e.*, urates make up a portion of the deposit.

A significant feature is that in the irritative and inflammatory stages of the disease, except in those where pus forms, if the patient receive vigorous anti-gout treatment, the dental inflammation subsides.

While pericemental irritation may involve many teeth, acute outbreaks are usually confined to but one or, at most, two teeth. The disease subsequently attacks other teeth singly, although these may escape involvement for years.

Diagnosis.—The symptoms and clinical history given clearly differentiate this condition from the pyorrhœa due to subgingival deposits and phagedenic pericementitis. When, however, the discharge of pus at the gum-margin occurs the case may be indistinguishable from phagedenic pericementitis; the difficulty of differentiation is all the more increased from the fact that both occur in patients affected by the same classes of general disorders. In phagedenic pericementitis, however, some local explanation of the disease-process may be elicited; in gouty pyorrhœa the symptoms may arise and the disease progress, frequently to its end, without any evident local sources of irritation, except that in some cases, sources of pericemental debility exist in faults of occlusion, leading to overuse, disuse, or misuse of the teeth.

Mode of Action of the Gouty Poison.—The conditions called gouty are held to be due to the retention in the circulating fluids of an excess of urates, a waste-product of tissue- and food-metabolism; this excess of material acts as an irritant and inflammation-exciting agent in the tissues of the body, producing alterations of function and structure in many tissues and organs, but most palpably in the members of the con-

nective-tissue group. The association of an excess of urates with gout was demonstrated by Garrod, who detected crystals of urates in the serum of blisters from gouty patients. The association became still more clear after an examination of the calculi of gout, which were found to contain urates. In gouty joint-affections urates of sodium are found in the diseased areas.

Uric acid belongs in the group of animal poisons generated in the living tissues of the body, to the general class of leucomaïns. It is an oxidation-product of albuminous matter. There is a series of these substances formed in the body, each representing a degree of albuminous decomposition by oxidation. The first and least oxidized, is hypoxanthin; second, xanthin; third, uric acid; and, fourth, urea, a substance freely soluble in blood-serum, which is excreted by the normal kidneys as a product of the nitrogenous waste of the body. That uric acid is formed instead of a corresponding amount of urea in conditions of faulty oxidation, is the general opinion of pathologists of the present day, an opinion not entirely demonstrable, as criticism [1] will show; still, it furnishes the only tangible explanation of the conditions of its formation and action at present available. Uric acid exists in the blood-serum as a quad-urate of sodium and magnesium. Sodium bi-urate is relatively a very insoluble material; if the sodium be displaced by lithium or potassium, the urates of these metals are formed, which have a greater degree of solubility.

If for any reason, notably disease of the kidney, the excretion of the urates be interfered with, they accumulate in the circulating fluids, causing an excess of urates without an increased production. Its increase may, on the other hand, be due to increased formation of uric acid. This is observed following upon the ingestion of unusual amounts of nitrogenous foods. After the consumption of malt liquors and sweet wines, particularly champagne, an increase of uric acid is observed, showing that food-metabolism as well as tissue-metabolism is concerned in the production of its excess. An increased production is also noted in conditions where there is an increased production of leucocytes and a diminution of the red corpuscles—the oxygen-carriers—so that the formation may be clearly traceable to a deficiency of oxygen carried to the tissues.

Another point to be noted is that an accumulation of waste-products, even a slowing of the lymph-flow about cells, interferes seriously with oxidation, even though oxygen be present in normal amount. The existence of these conditions, which cause sluggish vascular flow to and from tissues, must, therefore, be regarded as an important factor in the presence of an excess of the products of insufficient oxidation.

[1] Levison, *Gout*, 1896; Luff, Croonian Lectures, 1897.

Under some conditions deposits of crystals of urates occur in the connective tissues of the body, mainly those in which the circulation and the nutritive currents are sluggish, as in articular cartilages, usually of small joints; that is, the deposits occur in parts not freely flushed by the movements of intercellular fluids.

Ebstein believes that local tissue-changes precede and determine the point of attack and deposition. His opinion, as originally set forth,[1] was that coagulation-necrosis of the cells of a part occurred, and the tissues acquired an acid reaction, which determined the precipitation of urates in the area.

Van Noorden[2] believes that the point of deposition is determined by the local formation of a ferment, and that the deposition is independent of an excess of urates in the circulation.

A predisposition to degenerative changes exists in the tissues of gouty patients, notably in the arteries, leading to atheromatous changes, causing increased arterial rigidity. These changes are in part explainable by the presence of an excess of the waste-product—urates. Certainly in gouty patients, before deposits of urates occur and cause their characteristic effects, changes in many fibrous tissues are observed which are only explainable by constant irritation. Fully developed gout represents a degenerative and partially necrotic disease; but antedating these changes, it appears that there is an irritative, perhaps preceded by a stimulative stage.

Increase of connective tissue, as in atheroma of arteries, beneath mucous membranes and other situations is found in gouty patients before any history of acute outbreaks is noted.

Such functional disorders as neuralgia, no doubt due to anatomical changes, are also observed. All of these point to a long-continued period of irritation leading to an increased formation of connective tissue. A corollary of this state is a diminished vascularity, followed by diminished nutrition, hence debility of the part affected.

Given, then, a cause of tissue-debility, such as overuse or disuse of a part, with an accumulation of the waste-product, urates, the deposition of these salts is probable in the ill-nourished and debilitated tissue or structure, such as small joints. It will be recalled that the metatarsophalangeal joint, from its anatomical situation, is one of the joints of the body subjected to the greatest use; and lack of normal exercise of it (disuse) would be followed by its debility, and determine in a gouty patient the deposition of urates in it. These deposits occur first in the least vascular part, upon the surfaces of the articular cartilage, and excite irritation; if present in sufficient amount, inflammation of the surrounding vascular parts is aroused, and necrosis of the tissues which

[1] Flint's *Practice of Medicine*, 6th ed. [2] *Medical News*, Nov., 1895; and Ziegler.

are the seat of the deposit occurs. This constitutes the condition of acute gouty outbreak. Any of the joints may be attacked, although usually it is one of the small joints, most frequently the metatarso-phalangeal.

Gout, or uric-acid poisoning, may exist as a chronic affection without acute outbreaks; deposits accumulate in small joints (tophi), as of the fingers, causing stiffness and deformity, as the joints are successively affected. An injury to a joint may determine the affection in that joint, and any joint may be affected (Flint).

Gout may exist as an obscure affection without any of the joint-affections noted. Disorders of the stomach, liver, kidneys, heart, blood-vessels, and lungs may all attend chronic gout, and be caused by it. The evidence of connection of obscure conditions, such as headache, hebetude of mind, lassitude, digestive, circulatory, or respiratory troubles, with the gouty condition may only be made manifest by their relief through anti-gout therapeusis.

All forms of gout are largely hereditary. The manifestation of the diathesis may skip one generation and appear in the next. Hereditary gout in the female may manifest itself as rheumatoid arthritis. In a proportion of cases no heredity can be traced, although the existence of gout in the individual is unmistakable.

The deposits in gout are only readily detected when they exist as defined concretions. They may be present as fine crystals and escape detection.

Pathology.—The test of the soundness of the theory that there are distinctive gouty dental affections depends upon whether their causation, pathology, and symptoms are explainable by the phenomena of gout exhibited in other parts, and, again, by the effects of anti-gout therapeusis.

First, an examination of the teeth themselves. Teeth lost through the disease whose symptoms and clinical history have been given present the exterior appearance before described. Upon section enamel, dentin, and cementum are found to be highly organized. The pulp-chambers are frequently almost obliterated, even without external evidences of abrasion or erosion. Data relative to the condition of the pulp and pericementum are wanting, although from the degree of immobility of the teeth, it may be inferred that the pericementum is markedly diminished in volume prior to the beginning of the disease.

A tooth lost through this affection has, as stated, a degree of root-denudation—pericemental necrosis—governed by the stage of disease at which the tooth was extracted. Calculi found in the necrosed area give the reaction of urates, masked by the presence of calcium-phosphate deposits.

Without the existence of any of the local causes of acute pericementitis, the symptoms of this disease arise; they may subside, and leave the pericementum permanently crippled, or they may cause necrosis of more or less tissue and then subside. The same tooth is liable to succeeding attacks until the destruction of pericementum is complete.

Local therapeusis alleviates, but does not cure the condition. In its earliest stages anti-gout therapeusis affords marked relief. Are these phenomena explicable by the pathogenesis of gout? Acute and chronic gout may persist for years in joints of gouty patients, and yet the pericementum escape; again, the dental disease may exist and no history of gout, hereditary or acquired, be elicited.

Dental hyperæsthesia, a disposition to grind the teeth at night, shifting of positions of the teeth, and abrasion have all been noted as accompaniments of gout by Graves, Duckworth, Bartholow, Garretson, and others. All of these factors admit of explanation upon the theory of a gouty causation.

First, as to the character of the teeth. The high degree of organization of the teeth—*i. e.*, the formation of intercellular substance, formed matter of dentin—is explained upon the hypothesis of it being similar to sclerotic changes in other connective tissues. It is inferred that the soft tissues of the pulp and the pericementum are also involved in the process which leads to an increase of their fibrous elements. Doubtless, also, these changes involve the bloodvessels of these structures, lessening their calibre and their elasticity. This would represent the primary irritative stage of the uric-acid dyscrasia. Another expression of the irritative stage, altered glandular secretion, has been discussed under the head of erosion.

Why should gout attack the teeth (the pericementum) of some persons, and not those of others? and Why should it attack some teeth in preference to others? It must be remembered that the pericementum is anatomically a ligament as well as a periosteum, and that the union of a tooth with its alveolus is a joint. It is attacked for the same reason that any joint may be attacked: because it happens to be a weak articulation. The probable explanation of the selective action of gout-poison for the metatarso-phalangeal articulation is because this joint normally, in walking and standing, does a great deal of work, receives a blow and pressure with every step, and therefore would suffer early from disuse, according to a general physiological law. If it is not vigorously used, and it is not, in many gouty patients, its nutrition is disturbed and its vascular currents become sluggish. Analogous conditions may be established in the dental ligament, the pericementum. Owing to an increase in its tenuity and a decrease of its vascular

supply, its nutritive exchanges—its currents—become sluggish. If the teeth be used vigorously, the sluggish circulation may be partially counterbalanced; if not, vascular sluggishness is increased and a further predisposition to degenerations is established. Lack of mastication, a common failing in nearly all persons, is very common among gouty persons, particularly excessive eaters, who usually take food requiring little mastication. It is among this class of persons that gingivitis, accompanied by subgingival calculi, is common, producing the first variety of pyorrhœa, which may complicate or be aggravated by the local action of the gout-poison. The conditions of debility resulting from disuse of a structure exhibiting incipient degenerations, need but slight exciting causes to give rise to acute disease. The wedging of teeth, chance blows, and, among women, thread-biting, may determine an injury to the pericementum sufficient to make it the weak joint in which the gout-poison settles. In many cases a slight mal-occlusion may be the medium of injury to the dental articulation. This is most notable in those cases where chronic constructive changes in the pericementum have caused a thickening of some portion of the alveolar process between two teeth, causing the teeth to shift their position, and giving rise to the condition termed by Duckworth the "buck teeth" of gout. The teeth are brought into mal-occlusion, which determines the point of attack—the point of least resistance. These causes, both predisposing and exciting, may not be in evidence, in which event the teeth escape attack.

Given the predisposing and exciting causes which result in an area of lessened resistance in a portion of the pericementum of some tooth, when an excess of urates in the circulating fluids occurs, urates are deposited in the area of pericemental irritation; necrosis of the injured tissue, the seat of the deposit, occurs. Whether the deposit precedes necrosis, or the necrosis precedes the deposit, as originally set forth by Ebstein, is not known: an acid reaction is established which causes precipitation of urates. Inflammation is excited in the neighboring portions of the pericementum, which breaks down, as does also the adjacent alveolar process. An outlined or general swelling occurs, which may subside, or, if the inflammation persist, a discharge of glairy, mucus-like material may occur at the neck of the tooth, or in some cases pus—gouty abscess—discharges. The discharge may occur through the gum over the site of a circumscribed alveolar loss. As the gum-attachment is unbroken before the period of discharge, infection of the part has presumably occurred in the same manner as it occurs in cases of pulp-death without caries. The original uratic deposit afterward becomes obscured by deposits of the calculi which occur in continued pus-formation. These act as continuous irritants, so that the remainder of the pericemen-

tum and alveolar wall degenerates and disappears molecularly. In the absence of pus-formation the uratic deposits may be covered in or mixed with light phosphatic concretions, as in common tophi. In the absence of an apparent exciting cause for the location of the attack, it is presumed that the changes in the vessels have proceeded farther in some portion of the pericementum of one tooth than in others; the debility is most pronounced there in consequence.

The general pulpal hyperæsthesia noted in connection with the earliest stages of the disorder, may be attributed to the presence in the peripheral circulation of irritating material. The same explanation applies to the occurrence of attacks of pericemental hyperæsthesia, in which there is a disposition to grit the teeth. It will be observed that in both of these states there is early active hyperæmia of the parts. It is presumed that this is the antecedent and the cause of sclerotic changes which cause increased dentinal formation and increased density of alveolar walls, attended by increased tenuity of the pericementum.

Prognosis.—The prognosis of the disease depends largely upon the form in which it first exhibits itself, and also upon the length of time an increased amount of waste-products has been present in the circulation. In all grades of the inflammation, except that attended by pus-formation, the cure of the general condition is usually followed by a more or less prompt subsidence of the dental symptoms, although if a tooth be loose, the condition may improve, but does not disappear. If pus form, as indicated by the unusual activity of the inflammatory symptoms, destruction of tissue, pericemental and alveolar, it will progress until it is given vent, no matter how active or effective in other particulars anti-gout therapeusis may be. In these cases the tooth is usually lost sooner or later.

One or more teeth may be repeatedly attacked, and if the underlying cause be promptly removed, they may partially recover.

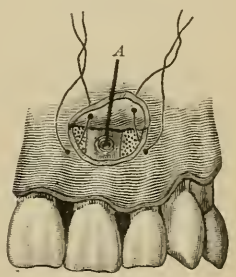
Because one tooth of a denture is affected it does not necessarily follow that others will become affected; a single tooth, or two teeth, may represent the weak articulations or points of selection of the gout-poison, and others remaining unaffected. It is usual, however, if the gouty condition be not held in abeyance, for successive teeth to become affected.

Treatment.—The treatment is both general and local; the importance of general therapeutics outweighing that of local measures of treatment.

The local treatment will depend upon the local conditions present. Some of these may be similar to those noted in connection with the previously described varieties of pyorrhœa—looseness of the teeth, mal-occlusion, infection, the presence of dead and foreign material, etc.

Each of these conditions requires correction by means already described. As regards the peculiar mode of infection and some of the anatomical conditions, the treatment in many respects is similar to that of phagedenic pericementitis. The site of actual infection, the situation of calculi and of dead tissue, the degenerating alveolar edges, and the unbroken gingival margin are similar, except that in the gouty cases the gingival attachment may be intact. It is even more important then in such cases that entrance to the area of necrosis be made through a special opening. A semicircular flap, as described in connection with the treatment of phagedenic pericementitis, should be raised and the disease-area explored and freely scraped to free it from all dead and dying tissue. The pockets should be syringed with hydrogen dioxid solution and touched with an antiseptic stimulant preparation; any of those previously given will answer (Fig. 377). The flap should next be stitched into place. An antiseptic mouth-wash should then be prescribed. In pyogenic cases finding vent at the gum-margin the treatment should be identical with that of phagedenic pericementitis.

Fig. 377.



A, calculus.

If the existing constitutional disorder do not receive correction, all local measures of treatment will be of but slight avail; not only may the disease appear upon other teeth, but the regenerative processes about the teeth which receive local treatment will entirely fail.

The treatment of the general condition concerns the general practitioner more than the dentist, but there are frequently attendant circumstances in these cases which render it not only advisable but imperative that the dentist should furnish direction as to diet and prescribe medicinally.

In many cases the evidence of a gouty condition, aside from the dental disease—and this is also true of dental erosion—are so faint that general practitioners, even if advised of the probable existence of masked or obscure gout, refuse to be guided by the indications pointed out and the diagnosis made by the dental practitioner. These dental signs will, no doubt, in the near future, come to be regarded by the general practitioner as valuable diagnostic indications of the existence of obscure gout; the evidences of dental induration, the existence of dental erosion, or the occurrence of pericemental degenerations, will all be regarded as pointing to the existence of some gouty disorder.

The general therapeutics of gout embraces medicinal agents and regulation of diet, the elimination of the gout-poison and the prevention of its formation. The principle of general therapeusis applied in the treatment of gouty conditions is diuresis. All agents which increase

secretion of urine have a beneficial effect. The ingestion of large quantities of water raises the blood-pressure and flushes the tissues of the body and the kidneys; hence elimination of formed waste-products is increased. If the water (as many "mineral waters") contains salts of potassium, the diuretic effect is increased. Solutions of potassium bitartrate (acid tartrate of potassium) are in general use for this purpose, forming the basis of many "fever mixtures," whose object is to rid the body of the products of nitrogenous waste. The citrates, tartrates, and acetates of potassium, sodium, and lithium, all neutral salts, when taken into the circulation are converted in the tissues into alkaline salts—carbonates—which render the urine alkaline, if present in sufficient amount, and increase excretion, provided they be taken in large volumes of water.

The danger from the presence of urates consists in the excessive formation of acid urates, the solubility of which is low. If converted into neutral urates, their solubility is increased and the tendency to deposition is correspondingly diminished. Upon this fact depends the efficacy of salts of lithium in conditions of gout. Taken into the circulation, neutral lithium urate is formed from the acid urates, an increased volume of which can be held in solution by the circulating fluids, the tendency to deposition being lessened. If the lithium salt be taken with free draughts of water, there is the additional factor of free diuresis. E. C. Kirk has introduced the bitartrate of lithium as a substitute for the citrate usually employed; its solvent power is greater and it has a specific diuretic action.

Increase of the alkalinity of the blood and the induction of free diuresis are the objects sought. It is by reason of their eliminant action that preparations of colchicum act as curative agents in acute gouty inflammations.

Piperazin ($C_4H_{10}N_2$, diethylene-diamin) is said to possess twelve times the solvent power for uric acid that lithium carbonate has. Hare,[1] however, failed to obtain any beneficial effects from the use of this drug in gout. Stewart[2] has recorded ill cerebral effects from its use.

The salicylate of sodium is commonly used in gouty affections as a uric-acid eliminant,[3] but more particularly to relieve attendant pains.

As the cases of acute gouty affections are usually readily recognized and treated by the general practitioner, the advice of the dental practitioner applies only in cases without classical symptoms; his recommendations, therefore, need scarcely exceed advising the free use of water containing potassium or lithium salts. A tablet of lithium bitartrate dissolved in a half pint or more of water, to be taken three times daily, is usually sufficient medicinal treatment.

[1] *Practical Therapeutics.* [2] *Ibid.* [3] Haig.

Advice should always be given as to a dietetic course which will minimize the formation of urates and secure elimination of the waste formed.

Measures should be advised to increase the oxidizing function. Free exercise in the open air is of first importance, and is only second to the correction of anæmic conditions; this, however, concerns the general practitioner, as it involves the prescribing of a course of iron or arsenic. Disorders of the intestinal tract and its appendages, particularly hepatic disorders, also demand correction.

The diet should be of a character which will lessen the formation of urates. The amount of vegetable food, in proportion to animal, should be increased, thus raising the alkalinity of the body-fluids. Red meats, and white meats difficult of digestion, increase the formation of urates. Poultry and shell-fish in the dietary lessen the formation of urates. The consumption of malt liquors notably increases it, and sweet wines, particularly champagnes (both sweet and dry), are poisonous to gouty patients. Spirituous liquors are also harmful, since they lessen tissue-oxidation and produce gastric and hepatic disturbances which cause faulty metabolism.

Recognizing the predisposition which exists in gouty persons to active pericemental degenerations, the operator should guard against injuries to the pericementum, which might induce a weak articulation and precipitate gouty pericementitis. Such teeth should not be wedged; injury to the gum or gum-margins, by the use of improper rubber-dam clamps, ligatures driven beneath margins, etc., may excite the first stages of a degeneration which will end only in the loss of the abused tooth.

SECTION VI.

CHAPTER XXVII.

DISEASES OF THE DECIDUOUS TEETH AND THEIR TREATMENT.

The deciduous teeth are subject to several diseases which affect the permanent teeth. Their crowns may be the seat of deposits—rarely, however, of salivary calculi. They may be affected by mechanical abrasion, dental caries, and acute diseases of the pulp, pericementum, and alveolar walls. In deciduous teeth septic pericementitis frequently runs a chronic course, but other chronic degenerations of the pericementum are rare, except in connection with constitutional diseases, notably rachitis.

Owing to peculiarities of structure and anatomical associations, diseases of the temporary teeth present features different from those of the same diseases occurring in the teeth of adults. The dentin of the deciduous teeth appears never to possess the high degrees of sensitivity noted in the adult teeth of some persons. In acute affections of the pulp the pains have less of a reflex character, being confined to the dental region; nor, so far as subjective phenomena are observed, are these pulp-pains of such severity as in adult teeth. The lymphatic connections of the deciduous teeth appear to be more free than those of the permanent teeth, so that involvement of the lymphatic glands—*i. e.*, evidence of septic absorption—is more frequently observed in connection with diseases of the deciduous than with those of the permanent teeth.

Acute catarrhal inflammations, including several forms of ulcerative action, belong so distinctively to the period during which the temporary teeth are in position that they are classified as diseases of the mouths of children.

All therapeutic measures, medicinal and mechanical, are directed toward insuring the non-septic retention of these teeth for a period of but five years, so that they may be and frequently are of a type differing from those directed toward the retention of dental organs for a lifetime; moreover, they are modified by peculiarities of anatomical relationships.

Loss of the deciduous teeth through other than physiological pro-

cesses should not be regarded with unconcern, for while it is true that pyogenic processes may exist for a long period upon the roots of a temporary tooth, its permanent successor underlying it being, at least so far as outward form is concerned, unaffected, it is more than probable that it is affected in its deeper histological structure and anatomical organization. The effects of too long retention and too early extraction of the deciduous teeth have been already discussed under the head of malpositions of the teeth.

Deposits upon the Teeth.

The deposits upon the deciduous teeth are usually confined to the cervical portion of the labial and buccal enamel, appearing as crescentic lines of green stain. These deposits, together with white deposits of débris in the gingivo-dental depression, are most frequently observed in the mouths of children whose teeth receive little or no care. They are probably due to the growth of chromogenic fungi in the remains of the enamel-cuticle, which persists longer in this situation than in any other. The only clinical significance, or rather pathological significance, of these deposits, is that they appear to furnish a predisposition to diffuse and ulcerative stomatitis; the connection between the two is by no means clear, although the association is frequent.

Such deposits are to be removed as described in Chapter XXIV. This trifling operation, as well as those of greater extent upon the deciduous teeth, is attended by difficulties absent in operations upon the teeth of adults. Children may be presented for dental treatment as early as the third or fourth year. At this age they are too young to appreciate the importance of dental service, or to be reasoned with; difficulties which are still further increased by insubordination, frequently abetted rather than curbed by the child's guardian. Children will rarely submit to inconvenience, much less to any degree of suffering incidental to dental operations. The physical character of the infantile mouth furnishes additional obstructions: the entrance to the mouth is small; the mouth-cavity is shallow owing to the shortness of the teeth and the alveolar process; and the salivary secretion is very free; thus dryness of the parts, one of the most efficient aids to accurate and painless dental operations, is but imperfectly attainable. It is rare that the rubber-dam can be used before the sixth or seventh year. The muscles of the tongue and lips of children are singularly uncontrollable.

Much may be accomplished with children through tact upon the part of the operator. Until the child has become familiarized with the dental chair, and with having its mouth examined, teeth touched, pressed upon, etc., operations should be confined to trifling procedures occupying but few minutes at most. The growing familiarity will

secure quiet of the patient, the most essential preliminary to operating. Lack of patience, or the use of force upon the part of the operator, is, except in rare cases, an effectual bar to present or any future operations. Many children may be given a permanent dread of dentists and dental operations by some ill-considered move upon the part of the operator.

Even premising quiet of the patient, the form of the mouth, lack of dryness, and necessity for short sittings make operations on the deciduous teeth a compromise between what should be done and what can be done.

It is quite as important that the temporary masticating apparatus should be kept in full working condition as it is for the permanent. As emphasized by Guilford,[1] during the period that the deciduous teeth are in position the alimentary canal and its glandular appendages are still in a developmental stage, and any disturbance of oral physiology, resulting in the stomach receiving poorly masticated food-stuffs, may react upon its normal development. Such cases may at least induce functional gastro-intestinal disturbances of some gravity. The existence of painful diseases of the teeth materially hinders mastication, so that their correction is demanded to relieve pain and to restore the partially lost function.

Abrasion of the Deciduous Teeth.

The occlusal surfaces of the deciduous teeth may wear away to a great extent. In some cases, this appears to be due to active fermentative changes in the mouth, which cause a general acid reaction of the fluid contents of the mouth; owing to faulty organization of the cusp-enamel, it may be readily soluble, and the teeth may be worn down and abraded through the combined action of solvents and the mechanical abrasion of mastication.

The cases of this condition observed, while associated with the presence of dental caries in some of the teeth, showed an exemption from caries in the teeth most abraded—those which were washed clean. More than this, in some of the teeth, worn nearly to the gum-margin, evidences of secondary constructive action upon the part of the pulp appeared; the teeth were worn beyond the original limits of the pulp-chamber and the pulps were still vital.

Another form of abrasion may be seen in teeth whose tissue-organization is not faulty. Children whose rectums are infested with parasites, as the ascaris lumbricoides, tænia (tapeworm), etc., and who suffer from irritable bladder due to hyperacidity of the urine, commonly have a reflex stimulation of the muscles of mastication during

[1] *Proc. Academy of Stomatology*, 1896.

sleep, which causes the forcible grinding of the teeth. The enamel of the teeth may be worn down and abraded, as observed in adults.

Treatment.—The treatment of abraded surfaces where the dentin is exposed is to wash the mouth with an antiseptic, not one containing chlorids, but hydrogen dioxid. Dry the dentin-surfaces and rub them vigorously with fused silver nitrate. This silver salt, rubbed upon dentin, forms with its organic constituents an albuminate of silver, which is persistently antiseptic. Under the influence of light it is reduced to the oxid of silver, which is slowly converted into silver lactate through the lactic acid produced in the mouth. Silver lactate, as contemporary surgical practice testifies, is a most efficient antiseptic. Contrary to previously held opinions, Truman's experiments[1] indicate that silver nitrate is a very penetrating coagulant, so that the reactions above given persist for a long period. It is in consequence of these peculiarities that silver nitrate is found so effective in checking and preventing dental caries.

The continued use of antiseptic mouth-washes is advised. Listerine, to which a minute portion of saccharin has been added to sweeten it, diluted one-half, is an agreeable antiseptic for continued use.

In cases of grinding of the teeth, the source of the reflex disturbance should be removed by the general practitioner. Belladonna, which children stand well relatively large doses of, is the medicinal agent most used to lessen vesical irritability. The urine, however, must have its acidity lessened through an increased vegetable diet, and, if required, potassium salts. Rectal parasites should be removed through the use of vermifuges—santonin, male fern, or others, depending upon which parasite is present. Small seat-worms, causing pruritus, may be destroyed by rectal injections of weak solutions of phénol sodique, a teaspoonful in a half pint of water.

Caries of the Deciduous Teeth.

Unless constant supervision of the deciduous teeth be exercised, caries is very liable to progress to the extent of pulp-exposure without the previous warning sign—dentinal sensitivity.

While the canal-portions of the pulps of these teeth are frequently fine, flattened, and tortuous, the pulp-chamber has a relatively large size, so that pulp-exposure follows quickly upon loss of a section of enamel. Eternal vigilance is the price of pulp-salvation in the deciduous teeth.

The approximal surfaces of the deciduous teeth appear to be more quickly and generally affected than the occlusal surfaces. Again, the occlusal surfaces of the second molars are more frequently affected than those of the first molars. The anatomical forms and arrangement of

[1] *Proc. Academy of Stomatology*, 1895.

the teeth explain these conditions; the approximal surfaces particularly of the molars, and the occlusal surfaces of the second molars, afford lodgement for food-débris more readily than do other situations.

It is rare that decided pain, increased by applications of cold or heat, and excited by the presence of sugar in the mouth, occurs before actual exposure of the pulp. In a majority of the cases which present themselves throbbing pain, indicative of pulpitis, is present.

If cavities are observed before pain has been complained of, and prompt and quickly subsiding response to applications of cold water is obtained, indicating a normal pulp, the cavity should be excavated, with more regard to removing the marginal caries than to thorough excavation, and an application of hydrogen dioxid made. The dentin is dried, and an application of a 20 per cent. solution of silver nitrate is made for a few minutes, the cavity being subsequently filled.

In cases of adjoining approximal cavities there is a disposition for the affected teeth to press together and lessen the size of the dental arch. Bonwill advises as a practice, followed by uniformly good results in such cases, to cleanse the cavities (Fig. 378) and insert masses of pink gutta-percha base-plate. The constant biting upon the gutta-percha causes a separation of the teeth which increases the size of the arch and affords additional space for permanent successors. He advises that before the gutta-percha masses are inserted that small pieces of blotting-paper saturated with carbolic acid be laid against the dentinal walls and the gutta-percha be packed over them. The more efficient and persistent antiseptic silver nitrate may be applied instead of the carbolic acid. Kirk advises that asbestos-felt be heated to destroy any organic matter present in it which might combine with the silver, and then be soaked in a saturated solution of silver nitrate, dried, and kept in dark bottles away from the light. Small pieces of the prepared felt may be used as described.

FIG. 378.

Mode of preparing approximal cavities.

The silver-nitrate method is particularly applicable to shallow cavities in which excavation for filling is impracticable. The dentin-surface is cleansed and dried, and the fused silver nitrate is rubbed upon the surface. This may be done after the method of Craven: a platinum wire is dipped into the powdered salt and held over a flame until the powder fuses into a button. By this means applications can be directly and accurately made.

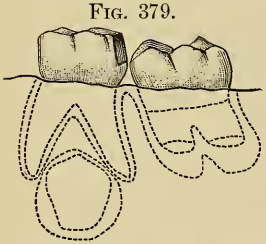
These shallow cavities frequently form upon the distal walls of second molars, and the erupting permanent first molar crowds into the carious area, not only reducing the space for the future bicuspids, but inducing caries in the mesial wall of the permanent tooth, and permit-

ting the deep invasion of, and pulp-destruction in the temporary tooth. If these cavities can be given a retentive form,[1] it should be done, and a filling having an exaggerated and rounded contour inserted, against which the erupting permanent tooth will press with a minimum of contact-area. In case of non-retentive form a disk is used to cut away the surface, leaving above the neck of the tooth a shoulder-like projection to hold back the permanent tooth (Fig. 379). The cut surface should be treated with silver nitrate.

Diseases of the Pulp.

If the case be seen at a stage when paroxysmal pain is caused by applications of cold, indicating active hyperæmia of the pulp, an attempt should by all means be made to soothe, protect, and maintain the vitality of the pulp. The principal object in maintaining vitality of the pulp is that the physiological process of root-resorption may not be aborted, prevented, or deranged. It is undoubtedly true that root-resorption does occur in the absence of a pulp, if the roots be in an aseptic condition ;[2] but in the absence of the pulp the process is irregular and incomplete (Fig. 380), and may not occur. Again, it is probable that the destruction of the pulp and the resulting imperfect resorption may disturb the nutritive balance in the developing alveolar structures in a manner at present unknown. The obtundent oils are of essential service in all of the pulp-disturbances of children. The oils of cloves and gaultheria are well-known domestic remedies for the toothache of children ; thymol is the most effective. The cavity of decay is syringed with tepid water and dried, and a pellet of cotton dipped in one of these oils is inserted. The cavity is given a retentive form, its walls painted with a non-conducting varnish, or, what is better, a pulp-cap filled with a paste (thymol, glycerin, and zinc oxid) is laid upon the deep wall of the cavity and a filling of zinc phosphate flowed over it.

Fig. 379.



Impingement of permanent molar upon the distal wall of the temporary second molar.

If evidences of active pulpitis—repeated paroxysms of pain, particularly throbbing pain—occur, it is advisable to destroy and remove the pulp. If soothed, and the cavity filled, the pulp dies and decomposes, and septic pericementitis arises. In any event, the pulp is first reduced to a condition of quiet.

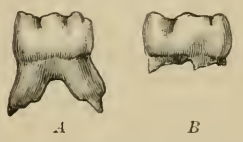
As to the means of destroying the pulp, it should be remembered that pulp-exposure, although it may occur at a very early age, usually occurs

[1] Woodward, *Proc. Academy of Stomatology*, 1896.

[2] *Proc. Academy of Stomatology*, 1896.

where the process of root-resorption has made some degree of advance; hence the communication between the pulp and apical tissues is more free than when the constriction of the apical foramen existed. It is evident, therefore, that the devitalizing agent (arsenic) must be used with extreme care, and in minimum amount, to prevent its passage into the apical circulation. The usual concomitant of the specific necrotic effects of arsenic upon adult pulps—jugulation of the pulp-circulation at the apical foramen—will probably not be in evidence because of the increased size of the foramen.

FIG. 380.



A B

A, root-resorption of pulpless tooth; *B*, normal resorption; vital pulp.

The usual shapes of cavities leading to pulp-exposure in temporary teeth demand that extra precautions be taken to prevent the escape of arsenic from the cavity upon the gum; moreover, that most useful adjunct, the rubber-dam, may not be applicable, so that the dryness of the cavity is imperfect. For these reasons the arsenic should be contained in devitalizing fibre. The amount used should be very minute and well diluted. The fibre is laid upon the exposure and retained by means of cotton and sandarac, or temporary stopping. If the evidences of acute pulp-disturbance have been very severe, it is advisable to retain the fibre by flowing over it thin zinc phosphate to avoid pressure. The application is not to remain more than twenty-four hours, and, if root-resorption have progressed, for not more than twelve hours. After this time the pulp-chamber is to be opened, when it may be found that the pulp has still a slight degree of sensitivity. Goddard[1] advises that glycerole of tannin be then sealed in the cavity for a week to tan the pulp.

Other means have been suggested and employed to effect pulp-destruction, to avoid the dangers incident to the use of arsenic. Darby uses with success a paste of about $\frac{1}{20}$ gr. of cantharides in carbolic acid. Dunbar[2] states that aqua ammoniæ applied to a pulp will effect its destruction. Tr. iodin has been advised by others. Increasing pressure by cotton pellets charged with oil of cloves is a slow method of destroying a pulp.[3]

The pulp is removed as from the adult teeth, any living filaments being destroyed by a drop of trichloracetic acid. Previous to opening the canals the mouth should be drenched with antiseptics, and the carious cavity be repeatedly washed with pyrozone. Dryness should be maintained as well as possible, and the rubber-dam used wherever it can be applied. The cleansed canals are filled with an antiseptic oil, dried, and filled at once. Two materials offer themselves

[1] *American Text-book of Operative Dentistry.*

[2] Quoted by Goddard, *Ibid.*

[3] Flagg.

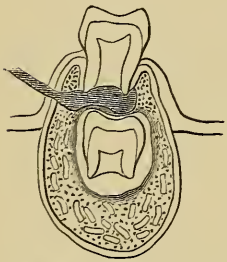
for this purpose in preference to all others—melted paraffin and balsamo del deserto. The paraffin may be combined with one of the iodin preparations, iodoform, aristol, or nosophen.

Septic Pericementitis.

Septic pericementitis presents itself as an acute or a chronic condition following upon death and putrescence of the pulp. It may arise in open cavities, or under fillings which were placed over dying pulps.

Its symptoms are those of septic apical pericementitis: the swelling is pronounced; pain is not so severe as in adult teeth, the tooth becomes very loose, and the duration of the disease is shorter than in the adult. The general symptoms, however, are frequently much more pronounced than in the adult; the inflammation may be attended by a chill, and frequently by a pronounced fever; the neighboring lymphatic glands may in a few hours exhibit evidence of the presence of septic matter in them, by swelling and tenderness. The point of exit of the pus is usually directly over the affected root, and not very far from the gum-margin (Fig. 381). If pus-exit be delayed, there is frequently a disposition to a stripping of the outer alveolar periosteum. This, in connection with wide spreading of the inflammation, is marked and common in strumous children.

Fig. 381.



Showing the relations of an abscess upon a temporary tooth with the crown of a developing permanent tooth underlying it.

Treatment.—The treatment consists in immediate evacuation of the pus and washing out the infected tract with antiseptics. The patient's head is steadied with the left hand, which also prevents the child seeing the bistoury. The last fingers are rested upon the teeth to prevent slipping of the knife through movement of the head of the child, and a direct cut is made to the process. The nozzle of a syringe charged with pyrozone is passed into the tooth-cavity and the fluid is driven through the abscess-tract. This is to be repeated until bubbling ceases. The cavity of decay is sealed after placing in it a pellet of cotton saturated with 2 per cent. formalin solution or similar disinfectant.

As soon as acute symptoms have subsided, an examination of the canals should be made to determine the amount of root-resorption. If this have progressed far, it will preclude the use of the more powerful germicides used in canal-cleansing. In any event, strong sodium dioxid can be used in the pulp-chamber, but its introduction into the canals must be guarded. Sterilization is effected there, by repeated washings with hydrogen dioxid or meditrina, followed by drying the canals and filling

them with cotton and an antiseptic oil—thyme or cassia—to test the thoroughness of disinfection. If the fistula heal and the tooth becomes tight, and the cotton when removed from the root has no odor of putrefaction, the canals should be filled with paraffin and aristol, or balsamo del deserto. In cases of high fever, where immediate access cannot be gained to the root-canals for disinfection, the immediate extraction of the tooth may be necessary.

The treatment of chronic abscess is similar to that of the acute variety after active inflammation has subsided. A longer time, however, is required for sterilization, as the dentin of the root is usually badly contaminated by noxious material and the soft tissues are in a state of debility, a condition identical with chronic infective ulcer. Sodium dioxid may be used with comparative freedom in these cases, as the passage of a small amount of the solution acts as a caustic to the diseased tissues beneath. Campho-phénique may be pumped into the canals of such cases without fear of ill-results.

If inflammatory symptoms run high, particularly in debilitated children, before root-resorption has progressed to any considerable extent, limited alveolar necrosis may occur, and a sequestrum be formed, which will require removal. In such cases, however, the tooth need not be extracted, if the sepsis can be controlled and the tooth be not markedly loose.

CHAPTER XXVIII.

REFLEX DISORDERS OF DENTAL ORIGIN.

Recognizing pain as a condition produced through the overexcitation of sensory nerves, a reflex pain may be defined as a pain referred to some point other than that of its origin. Pain referred to the distribution of a sensory nerve may be due to overexcitation of any portion of the nerve; in its terminal distribution; to diseases affecting any portion of the nerve-trunk, or to disorders affecting the central termination of the nerve. Again, irritation of one sensory nerve may be referred to some other sensory nerve. The condition is called neuralgia.

As both the upper and the lower teeth and their surroundings receive their neural supply from branches of the fifth pair of cranial nerves, discussion of this subject is confined to causes operating within the distribution of that nerve.

As such general conditions as malaria, syphilis, and forms of anæmia, operate to produce neuralgia which may be referred to the teeth, or the parts about them, only those cases will be regarded as dental which have undoubtedly a dental origin, as evidenced by disappearance of the neuralgia following cure of the exciting dental condition. It should be noted, however, that vague and sometimes severe pains referred to the teeth may entirely disappear after the cure of some constitutional disorder. For example, cases of periodically recurring dental pain have been entirely relieved through the administration of quinin and arsenic; the pains were clearly of malarial origin. Pain about the teeth in syphilitics has disappeared after the administration of iodids. Pain referred to the teeth in anæmic patients has disappeared after a long course of chalybeates.

Reflexes of dental origin are both motor and sensory, the latter far outweighing the former in importance. Motor reflexes may be noted in the quick spurt of saliva from the ducts of the salivary glands upon infliction of pain in the teeth, and by the spasmodic contraction of the muscles about the mouth when the pulp of a tooth is deliberately irritated. Twitching of the muscles about the face is a common accompaniment of trigeminal neuralgia.

Before direct association of dental diseases with pains in other parts can be clearly demonstrated a review of those conditions of the teeth attended by pain must be made.

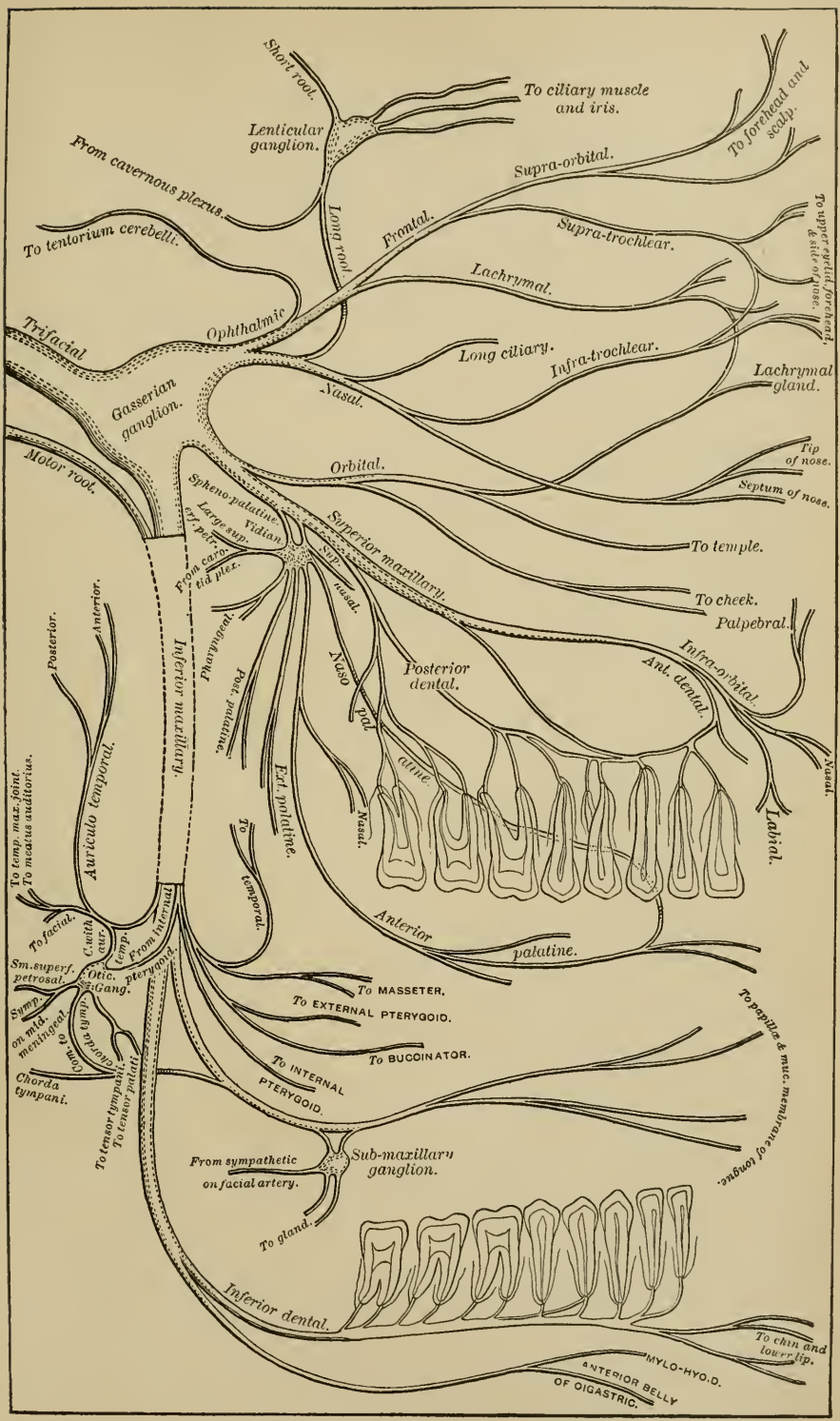


Fig. 382.—Plan of the fifth cranial nerve, showing the relationships of the dental nerves. (After Flower.)

Dental pain arises in consequence of disorder of the sensory structures; these are situated in the pulp, and by continuation throughout the dentin; and in the pericementum. The roots of teeth may have unusual anatomical relations with other sensory structures than their own pericementum. Dental pains, therefore, may be discussed, first, in connection with affections of the dentin and pulp, and, secondly, with those of the pericementum.

It was stated, in discussing the diseases of the dental pulp, that this organ is not the seat of the tactile sense, and that, like other organs having a kindred physiological relationship, irritation excited in it is not located, but is referred to some other part. While all reflex dental disturbances are, as a rule, located in some part of the great nerve-branch supplying the source of irritation, the irritation may be reflected to distant parts: first, of the same cranial nerve, and, secondly, to other nerves. That is, pain having its origin in one of the upper teeth is most likely to be referred to a point or points in the distribution of the superior maxillary nerve. Disturbances in or about the lower teeth are usually referred to the distribution of the inferior maxillary nerve. In affections of either upper or lower teeth the pain may be referred to the first division of the fifth nerve. In all of these cases, but most notably in connection with disturbances of the upper teeth, the usual symptom of trifacial neuralgia—tenderness of the supra- and infra-orbital nerves at their points of emergence upon the face, the supra- and infra-orbital foramina—is commonly present.

Cases are extremely rare where the reflex pain is referred to the opposite side; indeed, so unusual is this occurrence that its mention warrants suspicion that other sources of irritation exist upon the side referred to.

The extent or acuteness of reflex pain bears no direct relation to the apparent extent of the source of irritation.

As might be surmised from the function of the dental pulp, painful reflex dental disorders are more common in connection with diseases of the pulp than with those of the pericementum.

Reflex Neuralgia from Exposed Dentin.

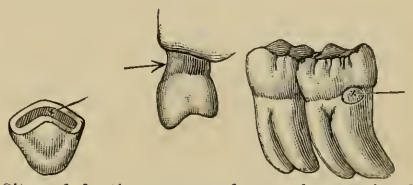
The exposure of the dentin to external sources of irritation is followed by reactions governed, first, by the degree of sensitivity inherent in the protoplasm of the tissue; and, secondly, by the degree of hypersensitivity induced in it. Reflex disturbance due to these irritations is more common in the class of women called "neuralgics" than in other persons. Like direct pulp-pains, unless actual pressure be exerted upon the affected tissue, there is no localized pain. In the absence of deliberate irritation, the pain may be referred to any portion

of the peripheral distribution of the fifth nerve upon the face; but if an acid liquid, such as lemon-juice or vinegar; or sugars, be taken into the mouth, pain is excited, which is referred indefinitely to the teeth of one side, frequently of one jaw. Reflex pains due to this cause are much more likely to appear when there is but little loss of dentin.

When carious cavities have proceeded to any depth evidences of direct pulp-disturbance are obtained through the increased response to thermal changes.

Reflex pains from exposed dentin appear most common in connection with exposures at the neck of the tooth and upon abraded areas. Obstinate and persistent neuralgia, positively referred to another nerve-branch, may apparently owe its origin to so slight a cause as exposure at the neck of a tooth (Fig. 383) of a line of dentin. The proof of the connection between the two is made clear by a disappearance of the neuralgia after the exposed dentin has been subjected to the action of powerful caustics, destroying the dentinal filaments to some depth. The connection between the two may be revealed only by accident; the contact of a tooth-pick, a dental instrument, or the finger-nail may induce a paroxysm of pain.

Fig. 383.



Sites of dentin exposure frequently associated with reflex pains.

While in some cases the dental origin of reflex pain may be made clear by the induction of a painful response in the area of reflection, by irritating a tooth-pulp, this reaction is not constant. The causal relation is only certain when the cure of localized dental disease is followed by a disappearance of the neuralgia without further treatment. This proof should be exacted in all cases.

The most common sources of neuralgic attacks about the face are diseases of the eyes and teeth. In general terms, diseases of the eye give rise to reflex pains referred to the distribution of the first branch of the fifth nerve; diseases of the teeth usually cause reflex pains in either the superior or inferior maxillary divisions, according as the upper or lower teeth are affected. In all painful affections of these nerves attention should at once be directed to the organs named.

Reflex Neuralgias from Pulp-diseases.

The disturbances require classification according to the distance between their source and their manifestations.

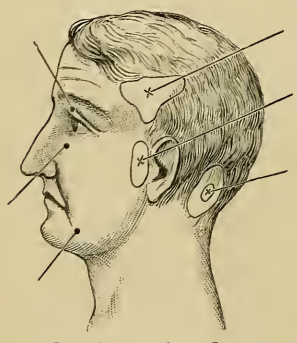
In the Fifth Pair of Nerves.—Pain referred to a different spot or area than its origin is a characteristic of all pulp-diseases. The extent of its reflection depends, first, upon the patient, as noted in con-

nection with the reflex pains from exposed dentin; and, secondly, upon the variety of pulp-disease. In neuralgic patients any variety of pulp-disease may cause comparatively distant pains. But, as Black has pointed out,[1] the general rule is, that the more chronic and profound degenerative diseases of the pulp are much more liable to give rise to distant reflex pains than are acute pulp-diseases.

The pains of acute hyperæmia and of acute inflammation of the pulp are usually referred to the region of the tooth affected, or to a corresponding nerve-trunk. In conditions of venous hyperæmia, nodular calcification, chronic inflammation, and, later pulp-degenerations, the pains may be of such character that their dental origin is only determined after persistent search. Particularly is this true of the growth of pulp-nodules. The source of the reflex pains is all the more obscure from the fact that in these chronic degenerations direct dental symptoms may be entirely absent, and are only elicited upon the most searching examination and exhaustive tests.

There is no constancy in the location of the pain due to any of these causes; but tenderness of the eyeball upon pressure; persistent pain in the temporal and anterior auricular regions, particularly in connection with pulp-diseases of the lower posterior teeth; in the ear itself, a common site of the reflex pain excited by chronic pulp-inflammation and suppuration of that organ; behind the ear, back of the lower border of the mastoid processes, tender spots may develop; tenderness to pressure may appear at the supra- and infra-orbital or mental foramen, and about the chin. In the same class of diseases the pains may frequently radiate as far as the shoulder. Many of these cases receive attention from the general practitioner, and the painful attacks recurring at irregular intervals are relieved by analgesic remedies—phenacetin, acetanilid, exalgin, etc.—and no attention paid to a probable dental source of the disorder. It should be a routine practice to examine the teeth in cases presenting pains of the type and in the situations described. Immediate search should be made for teeth containing pulps in late degenerative stages (see Symptoms of Disease of the Pulp). Acute diseases of the pulp, including suppuration and, notably, abscess of the pulp, usually have attention directed to the teeth through pain induced by thermal changes, so that their diagnosis is quickly made. Not so, however, with the chronic

FIG. 384.



Spots of tenderness in reflex neuralgias of dental origin.

[1] *American System of Dentistry*, vol. i.

degenerative changes, except possibly of pulp-nodules; for if the pulp is in the late stages of degeneration, it may require repeated applications of cold and heat to elicit a response from teeth which do not respond by tenderness upon percussion.

Failing to obtain evidence of pulp-disorders, examination should be made for exposed and hypersensitive dentin. Then, examination of the pericemental reaction of each tooth should be made and for any evidences about the teeth pointing to pericemental disturbance (see later).

Lauder Brunton[1] records that, in his own case, temporal neuralgia accompanied by tender eyeball was found due to exposed dentin upon the posterior cervical surface of a lower third molar (Fig. 383). The same writer[2] announces "that so frequently are headaches dependent upon decayed teeth that in all cases of headache the first thing I do is to carefully examine the teeth;" as should everyone else. Brunton explains the painful reaction upon the accepted hypothesis of the pathology of megrim, that it is due to spasmodic contraction of the peripheral end of an artery, with dilatation of the proximal portion. "Irritation in the tooth is reflected to the cervical sympathetic ganglia and causes spasmodic contraction of the arteries through irregular stimulation of the vasomotor nerves."

Reflex Pains from Diseases of the Pericementum.

As a general rule, pericemental pains are located at the affected tooth; but in some of the disorders, particularly those in which either hyperæmia or inflammation, acute or chronic, is not present, the teeth may not be tender upon percussion, and yet excite reflex pains in other parts, the proof of the connection being determined by a disappearance of the pain upon extraction of the tooth. The roots in such cases usually present either a hypertrophy of cementum, or show that resorption of a portion—it may be a major portion—of the root has occurred.

In cases of hypercementosis it is assumed that the source of the irritation is pressure upon the nerves of the pericementum by the hypertrophic growth. Very widespread disorders may arise from this source (see cases of insanity, etc.).

Flagg records[3] many varieties of trifacial neuralgia; pains in remote parts of the body; grave functional disorders of the eye and ear; and motor disturbances—chorea, epilepsy, and paralysis—having a direct demonstrable connection with hypercementosis. He mentions violent

[1] *St. Bartholomew's Hospital Reports*, vol. xix. Reprinted in his *Disorders of Digestion.*
[2] *Ibid.*
[3] *Dental Cosmos*, 1878.

attacks of trifacial neuralgia as the most common reflex disturbance from this source ; and next, long-continued pains in the ear or eye of the affected side. The existence of acute disease of these organs is usually diagnosed by the general practitioner. He states that oral and ocular disturbances, both functional and painful, are of gradually increasing severity.

In examining for a dental source of such pains, exposed dentin, pulp-diseases, and inflammatory affections of the pericementum should be first excluded. In examinations by percussion a different response may be obtained from some one tooth than from the others. Hypercementosis of a particular tooth is indicated by finding the gum-line slightly receded, and the tooth-attachment unusually firm ; if, in addition, vague, heavy dental pains have persisted at intervals over a long period, the diagnosis is probable. It is only certain when tapping upon the tooth brings on a paroxysm of neuralgia, or where a skiagraphic view actually exhibits the hypertrophic growth. The remedy is extraction. Any root-fragment left unextracted may perpetuate the reflex disorder.

Painful affections referred to the neighboring region of the affected tooth, or diffused through the distribution of the corresponding nerve-trunk, or to the eye or ear, may accompany the process of resorption of the roots of permanent teeth. Gillman[1] records a case where facial paralysis disappeared upon extraction of a tooth which had long been the seat of disturbances, and which upon extraction revealed resorption of its root.

All of the acute or chronic, septic or non-septic inflammations of the pericementum, may give rise to reflex pains. In many of these cases the cause of the reflex irritation is due, perhaps, to sepsis, rather than to a pure neurotic connection. The most common causes of the reflex pains are found in that stage of pericemental irritation which antedates acute septic apical pericementitis, and which accompanies the chronic inflammations of the apical pericementum from any cause. In some of these cases reflex neuralgia may play a subordinate part to general infection from the focus of disease. The reflex nervous disorders consist in painful disturbances in the distribution of the fifth nerve and disorders of special senses, particularly that of hearing. Unless an exacerbation of the reflex disorder, or symptoms referable to that region, be induced by pressure or percussion on the tooth, a causal relationship is only made out by either relieving an existing dental disorder or extracting the teeth. The symptoms of septic intoxication and septicæmia must be carefully differentiated from reflex neuralgias in such cases: the latter are rare ; the former probably more frequent than supposed in connection with septic dental diseases.

[1] *Boston Med. and Surg. Journal*, 1867.

Impacted Teeth as a Cause of Neuralgia.

Neuralgia of varying degrees of severity is a common accompaniment of impacted teeth. It is most frequently noted in connection with eruption of the lower third molars, not only because this tooth is the one most frequently impacted, but because of the anatomical relations of its roots with the inferior dental nerve.

In the milder forms of impaction, those in which eruption, though delayed, is subsequently completed, the pains are commonly localized and associated with but occasional attacks of rigidity of the masseter muscles. If, however, the crown present horizontally or nearly so, and its progress is arrested by impaction against the posterior wall of the lower molar, or if its progress be arrested by permanent imprisonment of the advancing crown between the posterior surface of the second molar and the base of the coronoid process, not only may intense local pains be induced, but severe reflex disturbances of both a sensory and motor character may occur. In some of these cases root-formation is completed, although the crown of the tooth does not advance, in which case compression of the inferior dental canal and its contents may occur and cause grave reflex disturbances. The local irritation about the root, due to root-growth, may excite continued constructive action by the pericementum, and the hypertrophic growth in its turn may be the source of reflex neuralgias.

Complete imprisonment of the entire tooth has been found to be the exciting cause of facial neuralgias, for the cure of which extensive surgical operations have been performed.

Impacted cuspids and other teeth may excite no other symptoms than reflex neuralgia. The possible connection between an impacted tooth and neuralgia is made out after excluding other dental causes, when it may be observed that one or more of the permanent teeth are absent from the dental arch, at dates long after their normal time of eruption.

A condition equivalent to partial impaction, in which dental irritation may be the source of reflex neuralgia, is seen when the teeth are crowded—jammed into arches too small for their accommodation. During the period of eruption severe maxillary pains may recur at intervals.

Pain referred to Nervous Tracts other than the Fifth.

The most common disturbance appearing in other cerebro-spinal nerves than the fifth, due to dental diseases, is an affection of the eighth or auditory nerve. Cases of deafness have been recorded due to diseases of both pulp and pericementum, notably to hypercementosis. Deafness which has persisted for a long period has been markedly lessened by the extraction of teeth the seat of disease. Cases of sup-

purative otitis media have been regarded as having pathological association with septic diseases about the teeth from the fact that the aural trouble subsided immediately after extraction of the diseased teeth.

Sensory disturbances of the eye, associated with dental diseases, have been alluded to; in addition to these, grave structural and functional diseases of the eye, traceable to dental causes, have been recorded, such as motor, sensory, and special sense-disturbances, together with trophic disorders.[1] Among the latter may be mentioned corneal inflammation and ulceration and phlyctenular conjunctivitis.

Irregular paralyses of the third, fourth, and sixth nerves of the affected side have been noted.

Amaurosis, amblyopia, and functional blindness without retinal conditions to account for it, have been found to arise from notably advanced degenerative changes in the dental pulp, sight returning to the eye after loss of a diseased tooth. De Witt[2] records a most instructive case where temporary blindness was associated with septic apical pericementitis, disappearing after evacuation of the abscess, and reappearing when secondary inflammatory action arose in the pericementum. The ocular affection disappeared permanently and almost entirely with the loss of the tooth. The history of this case illustrates the important causal relationship of reflex disturbances with late pulp-degenerations; for the blindness arose two months after some teeth were filled, and existed for twelve years before the septic apical pericementitis appeared.

A careful examination of these and all other reflex disturbances shows that pulp-degenerations outnumber all other affections as causes. Many or most of the cases are recorded by general practitioners, who make no distinction between diseases of the pulp and those of the pericementum, but a reliable diagnosis of the conditions is made possible by the accompanying descriptions.

Cases of ovarian and uterine neuralgia and sciatica and cases of obstinate pains in the toes and fingers have been traced to dental irritation of some one of the varieties named; the proof of association being disappearance of the pain with loss of the tooth.

Motor Disturbances from Dental Diseases.

Motor disturbances due to dental irritation may occur as recurrent or persistent contraction or paralysis of muscles, together with more or less general chorea; in rare instances epilepsy and hystero-epilepsy. Twitching of muscles of the affected side of the face, ranging from slight affection of the occipito-frontalis, to recurring spasm of the ele-

[1] See Brubaker, *American System of Dentistry*, vol. iii., for very full and detailed discussion of these subjects.

[2] Quoted by Brunton, *Disorders of Digestion.*

vators and depressors of the lower lip, are far from uncommon phenomena attendant upon pulp-diseases.

Contraction of the masseter muscle is a common accompaniment of retarded eruption of the lower third molar, which may be intensified until the condition is fitly termed trismus, in some cases of partial impaction of the teeth. Partial trismus has been found due to a general overcrowding of the dental arch.[1] Records of cases of torticollis, due to dental diseases, are also given by Brubaker.

Cases of facial paralysis, and cases of paralysis of one arm, of paraplegia and hemiplegia, and even of general paralysis, have been noted as disappearing after the extraction of diseased teeth. It is noteworthy that in these cases, as well as in several cases of tetanus recorded, the possibility of an infection entered into the pathogenesis of the nervous diseases.

Stellwagen[2] records a case where symptoms of partial hemiplegia followed upon the operation of capping the pulps of two molar teeth; the symptoms disappeared promptly upon extraction of these teeth.

Cases of insanity arising from dental diseases have been recorded; they were both maniacal and melancholic. In several of them a restoration to a normal mental state followed promptly upon removal of the offending teeth. In some of these cases a pre-existing maxillary neuralgia directed attention to the teeth as possible sources of the nervous diseases.

Dental Pain arising from other than Dental Sources.

Conditions of pain the reverse of those discussed—*i. e.*, pain definitely or indefinitely located in teeth which exhibit no morbid conditions whatever—demand occasional attention at the hands of the dentist. These painful states are most commonly found in gouty patients, in whom the pain may have the character of pulp-pains, or of pericementitis. The pains may recur at intervals, and be associated with headache, constipation, etc. The gouty origin of the pains is indicated by the efficacy of sodium salicylate in their treatment, without any dental treatment whatever.

Chronic malarial poisoning, as stated in the beginning of this chapter, may give rise to periodical attacks of maxillary neuralgia. As in the gouty cases, the constitutional cause of the disturbance is made clear through the therapeusis most effective, viz., the periodical recurrence of the pain leads to the inference of a malarial origin, and to the administration of quinin.

Syphilitic pains in the jaws have a pericemental character, and other evidences of syphilis are present which point to a diagnosis.

[1] Brubaker.

[2] Private communication.

Pains in or about the teeth are occasional accompaniments of diseases of the brain or its vessels, and of diseases of the uterus, kidneys, and bladder.

Disease in any portion of the fifth cranial nerve may be referred to the teeth.

Dental pain during pregnancy, without any direct evidence of dental disease, is relatively common.

Disorders of the lower bowels, causing constipation, may give rise to pain referred to one or more teeth, the pain ceasing promptly upon the administration of an active evacuant.

CHAPTER XXIX.

INFECTIONS OF AND FROM THE MOUTH, AND STERILIZATION.

The conditions found in the human mouth, as pointed out in Chapters III. and VI., are of a character which afford lodgement to, and opportunities for multiplication of, many forms of bacteria, both saprophytic and parasitic. The oral conditions are, however, not entirely constant, so that at different periods they may favor the development of some special bacterial forms more than others. The nature of these variations has not been made out, although their effects are indubitable. Again, the oral bacterial inhabitants are not constant as to species, for while there are many forms which appear to be invariable occupants of the oral cavity, many pathogenic forms are but accidental residents. Becoming resident, they may or may not develop according as they find in the mouth a suitable soil. The nature of what constitutes a suitable or unsuitable soil has not been determined, although in some cases extra-oral culture-experiments furnish some indications.

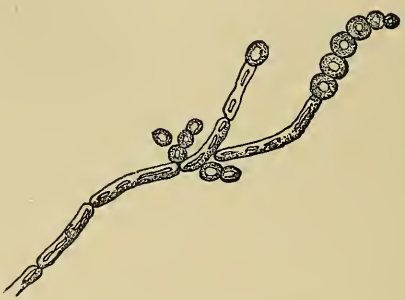
Bacterial growth, as causes of dental caries and diseases of the pulp and pericementum, have been discussed in connection with those several diseases. It was shown that the pyogenic cocci are almost constant inhabitants of the human mouth. There appeared also evidence that some of the reflex disorders of distant parts are directly traceable to septic processes about the teeth, and, in addition to these, suppurative diseases in other parts become curable after removal of a septic tooth; such conditions representing infection from a local dental infection, an important aspect of dental pathology.

The infections arising from the growth of mouth-fungi are local and general. The phrase fungi is used in this connection, because other classes beside the fission-fungi (schizomycetes) are pathogenic also. Both the thread-fungi (hyphomycetes) and bud-fungi (blastomycetes) induce morbid conditions in the human mouth.

The notable fungus of the blastomycetes, is the saccharomyces albicans; this organism, when classified by mycologists as a thread-fungus, was known as the oïdium albicans (Fig. 385). The growth of this organism illustrates forcibly the influence of soil on the growth of fungi. It does not occur in the mouths of healthy, well-nourished, and clean children with good surroundings. It is a disease of childhood, particu-

larly of nurslings, and its occurrence is almost always confined to bottle-fed babies whose feeding-bottles are kept in an unclean condition. De-

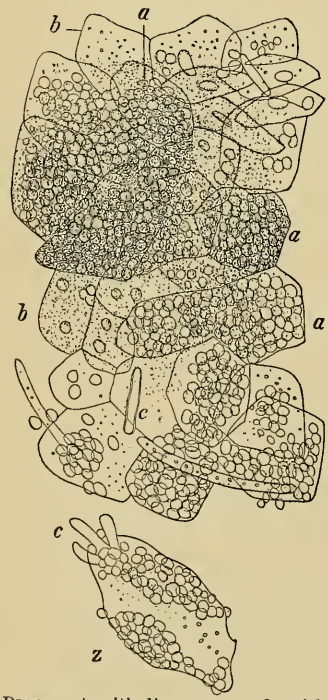
Fig. 385.



Saccharomyces albicans, thrush fungus. (Miller.)

bility of the oral tissues is established in consequence of the fermentations arising from the source named, furnishing a favorable condition for the development of the saccharomyces (oïdium) albicans. The condition produced is known as thrush. The infection may be carried from one child to another, and if the fungus be brought in contact with an abraded mucous surface of an adult it may develop.

Fig. 386.



Pavement-epithelium covered with spores of the *oïdium albicans*. (Ch. Robin.)

The fungus burrows between the epithelial cells of the mucous membrane (Fig. 386), not beyond it. It first appears in small spots, which coalesce, until large patches of a membranous-like growth cover extensive surfaces, spreading by continuity to all of the mucous surfaces associated with the mouth.

As bud-fungi flourish only in media of acid reaction, the use of alkaline washes is indicated in the treatment of this condition. Wiping the patches with dilute phénol sodique is also efficacious.

The hyphomycetes, or thread-fungi, although associated with diseases of the human skin, have not had any pathological significance attached to them as regards the mouth.

[1] Miller, *Micro-organisms of the Human Mouth.*

Infective Bacteria of the Mouth.

Bacteria, being ever present, must always play a part in either originating, modifying, or associating with all oral diseases.

That the progressive decomposition of albuminous substances, always present in the mouth to a greater or less degree, by the action of saprophytic fungi, must give rise to derivatives of albumin, many of them toxic in effects, would be surmised even in the absence of experimental demonstration, a suspicion confirmed by experiment. Vulpian[1] produced septicæmia by vaccinating animals with the saliva of a healthy man. Griffin[2] showed that the parotid saliva (pure) is harmless. The saliva, if boiled, exerts no toxic action, from which it is clear that it derives its toxic substances from the mouth. The saliva of individuals differs at times in the degree of its poisonous action. In some diseases it becomes intensely toxic.

Of the many oral bacterial forms, some are cultivable and some are not; hence the specific effects of some are discovered, others are doubtful.

With regard to local affections, other than those described in the body of this book, a bacterial causation has been made out in some, but in others it has not.

Stomatitis.

Definition.—By stomatitis is meant a catarrhal inflammation of the mucous membrane of the mouth.

Varieties.—It may be localized, as in marginal gingivitis, or be diffuse; and, again, be accompanied by localized tissue-destructions—ulcerations; the character of the ulceration differs according to its probable causes.

Occurrence.—Most of these diseases belong to the period of childhood, although localized ulcerative stomatitis may appear in the adult.

Causes.—The causes of stomatitis are so many and varied as to suggest a classification under heads according to assignable causes. While it is true that bacterial infection has not been shown to be a direct cause of all of these conditions, some degree of causal relationship is probable in all of them. The disease may, however, be included under two heads according as they are or are not localized, and necrotic. The less localized cases appear as a diffuse catarrhal affection, affecting wide areas of the oral mucous membrane; the others appear as spots of localized tissue-destruction attended by surrounding hyperæmia.

[1] Quoted by Miller. [2] *Ibid.*

- Catarrhal Stomatitis . . .
  - Local . . . .
    - Simple.
    - Infectious . . . .
      - Fermentations.
      - Diphtheria.
      - Gonorrhœa.
  - Symptomatic
    - Eruptive fevers.
    - Syphilis.
    - Tuberculosis.
    - Typhoid fever.
    - Drug-action . . .
      - Iodids.
      - Mercury.
      - Lead.
      - Pilocarpin.
- Ulcerative Stomatitis . . .
  - Local . . . .
    - Aphthæ.
    - Thrush.
    - Noma.
    - Herpes.
    - Syphilis (primary).
  - Symptomatic
    - Syphilis . . . . . .
      - Secondary.
      - Tertiary.
    - Tuberculosis (local).

SIMPLE LOCAL CATARRHAL STOMATITIS.

The general symptoms of catarrhal inflammation—heat and swelling, with deepened color of the mucous membrane, followed by increased secretion and exudation—attend several types of oral irritation, such as the irritation induced by erupting teeth, particularly of the deciduous teeth. Inflammation of any degree may follow the taking into the mouth of caustic chemical substances, such as caustic alkalies, mineral acids, carbolic acid, etc., which are occasionally taken by children. Other irritant drugs, and very hot fluids may produce similar results. General catarrhal stomatitis is a frequent affection of confirmed smokers, and of drinkers of distilled liquors.

The cure of these conditions consists in the removal or neutralization of the cause, and the use of local sedatives and antiseptics to allay irritation and prevent infection. The most effective method of treating the inflammatory condition is by antiseptic sprays, such as diluted Dobell's solution, followed by sprays of strong solutions of potassium chlorate. If much pain exist, phénol sodique is an admirable sedative antiseptic, used in 10–20 per cent. solution, as a spray.

INFECTIVE LOCAL CATARRHAL STOMATITIS.

This in some degree is a common, perhaps the necessary, antecedent condition to many of the ulcerative forms of stomatitis. It is probable

that many of the cases of stomatitis found in infants, children, and adults are due to unusual fermentations occurring in the mouth. Children whose nursing-bottles are not kept clean; those who at a later age suffer from neglect of the teeth and from the effects of improper food; adults in whose mouths dental diseases are widespread, and whose oral hygiene is very faulty: all exhibit abnormal conditions of the oral mucous membrane—more or less swelling, softness, and deepened color of the mucous membrane, a coated tongue, and offensive breath, with an increase of oral secretions.

The complexus of oral symptoms is commonly, and also by the general practitioner, regarded as symptomatic of gastric, intestinal, and hepatic disorders, as doubtless they are, but the causal relationship is in many cases probably the reverse of that implied in such opinions, for it is probable (see later) that the disturbances of digestion are fermentative in character, and the organisms causing them find their way to the stomach from the mouth, which was first affected. The treatment of this condition consists in the correction of its causes, their non-repetition, and the continued use of oral antiseptics.

While the point of first attack of the diphtheria bacillus is most marked about the soft palate and tonsils, the false membrane forming there and spreading to the pharynx, more or less general inflammation of the oral mucous membrane also occurs. The gonococcus may be lodged in some portion of the oral cavity and excite its specific effects upon contiguous mucous membranes.

SYMPTOMATIC CATARRHAL STOMATITIS.

Stomatitis in its catarrhal form is usually associated with the early and later stages of the eruptive fevers, scarlet fever, smallpox, etc. In scarlet fever and smallpox evidences of direct infection of the mouth exist and the inflammatory reaction is pronounced.

Catarrhal stomatitis is one of the manifestations of secondary and tertiary syphilis, antedating the appearance of tissue-necrosis (ulcerations).

More or less catarrhal stomatitis, confined, it may be, to the mucous membrane of the gums, is common in the mouths of phthisical patients; this condition exhibits no evidence of direct association of the local development of the bacillus of tuberculosis, because no tubercular ulcers may arise or threaten.

The stomatitis of typhoid fever may be regarded as an almost essential feature of the disease.

The effects of drug-elimination by the oral tissues have been already discussed (see Chapter XXIV.).

ULCERATIVE STOMATITIS.

It has been customary to describe ulcerative stomatitis as simple and infective; in all probability these ulcerations are always infective. Like catarrhal stomatitis the ulcerative disease may have only a local significance or be indicative of some general disease.

Ulcerative Stomatitis of Local Significance.—The more usual or infantile forms of these disorders are a sequel of catarrhal stomatitis, at least of an acquired debility of the oral tissues, and their primary cause is, therefore, the cause producing a condition of mucous membrane which permits the growth of infective organisms. One of these diseases, thrush, has already been described. The others, aphthæ, herpes labialis, and noma, are all probably due to the action of organisms.

Aphthæ.—This affection is common in its isolated form, as the canker sore. In the catarrhal stomatitis of children, during or after dentition, multiple sores frequently make their appearance. The condition can best be studied when it appears as an isolated sore in the mouth of the adult. The most common situation of the sore is at the junction of two mucous surfaces, such as that of the gum with the lip or cheek, or that of the floor of the mouth with the gum or tongue. Redness diffused over a limited area, followed by a nodular hardening, occurs, during which local pain is annoying; the centre of the hardened area breaks, forming a raw surface, which quickly acquires a rough white coating which is easily removable. The sores are very painful.

This condition follows so constantly upon the taking of very indigestible food, such as lobster, Welsh rarebits, etc., that acute indigestion must be regarded as having some causal relationship with it. It is also of frequent occurrence in the mouths of dyspeptics; that form of gastric disturbance attended by a deficiency of hydrochloric acid in the gastric juice appears to have a constant association with it.

The appearance of ulcerative stomatitis in children, together with its treatment, was discussed in the chapter on Dentition.

The general treatment of these ulcerations appearing in the mouths of children is the administration of a laxative, and the subsequent administration of listerine, gtt. x, every two hours. Locally the mucous membrane is to be sprayed with pyrozone, followed by sprays of strong solutions of potassium chlorate.

Localized aphthous patches in the adult are promptly relieved by the administration of calomel, gr. ij at night, followed in the morning by a mild saline. The local sore is dried and touched with pure carbolic acid. The administration of alkalies before meals, and hydrochloric acid after meals, usually remedies the gastric condition, unless it be of long standing.

A variety of aphthous sore is called, from the anatomical situation of the ulcers, follicular stomatitis. Irritation and swelling of the mucous follicles in the palatal, buccal, and labial mucous membrane are accompanied by more or less localized inflammation; the follicles become ulcerous, the small ulcers having a uniform size. This condition quickly disappears under the treatment advised for ulcerative stomatitis. An indication of the bacterial origin of all of these disturbances is seen in the efficacy of antiseptics used in their treatment.

Noma, Cancrum Oris, Gangrene of the Mouth.—In ill-fed, ill-nourished, and ill-kept, cachectic children, the debilitation of the oral tissues may exceed the grades given, and a disease probably bacterial in origin may arise which leads to widespread necrosis of the cheeks and maxillæ. The condition is called gangrene of the mouth, noma, or cancrum oris; the latter term has been applied to the less severe varieties.

This disease may make its appearance as an ulcer at the junction of cheek and gum; in other cases a severe stomatitis arises without a primary ulcer. A greater or less extent of the cheek acquires a board-like hardness, becoming livid; the overlying mucous membrane breaks,

Fig. 387.

Noma. (J. Lewis Smith.)

exhibiting a large slough. The necrosis extends toward cheek and jaw, destroying further tissue. The sloughs undergo putrefactive decomposition, emitting a stench. The destruction of tissue may be arrested, or may proceed, destroying in a few days the entire cheek and bony

tissues. In the more severe cases the disease is almost invariably fatal, because the extent of the tissue-destruction bears a constant relation to the underlying debility of the patient. It will be seen that the disease resembles malignant pustule or carbuncle in several of its features.

While no specific organism has been isolated as pathogenic of this condition, Schimmelbosch[1] found a bacillus (pure culture) upon the borders of the necrosis, which may prove pathogenic of noma.

These cases are purely medical, so that their full discussion is not warranted in these pages. The principle of treatment is to improve the general condition of the child, destroy the probable infection in the borders of the still vital tissue, and promote sloughing of the necrosed tissue.

Syphilitic Affections of the Mouth.—The recognition of syphilitic lesions about the mouth is of vital importance to the dental operator, first, because by the recognition he may take steps to prevent the carriage of infection to innocent patients; and, secondly, that he may avoid inoculation of himself by the poison.

In the minds of many, syphilis is associated with the lower class of persons, who are confirmed *débauchés*. While it is undoubtedly true that its prevalence is most marked in this class of persons, it appears, and with horrible frequence, in persons who would be little suspected of having such infection. The operator is to be guided in his opinions and precautions in this matter, not by the social status of the patient, but by the nature of the morbid conditions existing.

Syphilis is usually divided into three stages, primary, secondary, and tertiary; to these may be added a fourth stage, viz., in patients who have been discharged as cured mild manifestations of disorders, particularly of the skin and mucous membranes, make their appearance from time to time, and disappear promptly upon the administration of iodids.

The first stage of syphilis—primary syphilis—consists in the formation of the primary sore or chancre, and the involvement of the nearest lymphatic glands. Secondary syphilis is attended by fever, eruptive inflammations of the skin, inflammation and superficial ulcerations of mucous structures. In tertiary syphilis destructive inflammation of the skin, mucous membranes, and connective tissues occurs, together with the formation of specific tumors—gummata.

Some difference of opinion exists among syphilographers as to the relative infective power of the secretions from the several lesions of syphilis. All are agreed, however, that the secretions from the secondary lesions observed in and about the mouth are highly infective. It is the part of prudence to regard all syphilitic lesions as infective. All

[1] Miller, *Dental Cosmos*, Sept., 1891.

these stages of syphilis may be seen in the human mouth. It is to be remembered that if the mucous membrane of the mouth be infected from a mucous patch (a secondary lesion), the acquired disease will appear, not as a mucous patch, but as a chancre. It is from mucous patches that infection is most to be feared.

PRIMARY SYPHILIS OF THE MOUTH.—*Causes.*—The primary lesion of syphilis, chancre, when found in the mouth is a consequence of direct infection from a syphilitic. The infection occurs from contact of the mucous surface of the mouth with a syphilitic lesion upon another person: it has been transmitted by kissing: it may occur from using a glass or cup previously used by a syphilitic, by smoking cigars or cigarettes which have been made by syphilitic cigarmakers, who have applied the tongue to the tobacco in attaching the wrapper. Any of the articles named, or the contact of any article which has been in contact with a syphilitic lesion, if brought in contact with an abraded mucous surface may cause infection.

The infection may be transferred from patient to operator if the fingers have any abraded surface, or if the surface is broken accidentally by an instrument. Infection may be transmitted from one patient to another by any instrument, appliance, or article used by the syphilitic being afterward used by an innocent person. Drinking-glasses, mouth-mirrors, exploring-instruments, rubber-dam, rubber-dam clamps, saliva ejector tubes, lancets, forceps, or any other instruments may be the medium of communication. During and since the time of Hunter the use of teeth from syphilitic patients in plantation operations has been a clearly recognized medium of communication.

Appearances and Diagnosis—"The primary lesion of syphilis never makes its appearance before ten days after infection; the maximum period is about ninety days; the average is twenty-one days."[1]

It usually appears as a single, elevated, hard papule. In cases of dental infection, most frequently about the lips, the papule loses its epithelial coating after some days. The induration surrounding the papular mass increases until the papule, which is now raw and in a process of ulceration, appears surrounded by a ring of cartilaginous hardness. This induration is the one distinguishing feature of the chancre, which is not painful. In about a week after the appearance of the primary sore, swelling of the submaxillary lymphatic glands is observed. In case the chancre appear upon the tongue, the subhyoid lymphatic glands are swollen.[2] Unless pyogenic infection have occurred, the lymphatic involvement is not inflammatory, there being no pain present. In from three to four weeks the sore disappears, leaving no signs of its site in some cases; in others, some induration may persist.

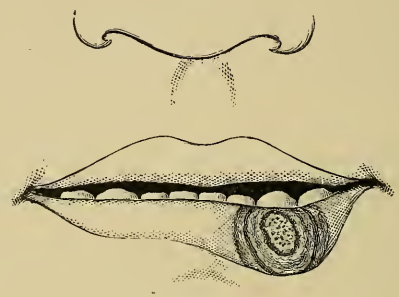
[1] Gross, *System of Surgery.*

[2] Park, *Surgery.*

The diagnosis of this condition is the important consideration, so far as the dental practitioner is concerned, its treatment being the province of the general surgeon.

The elevation of the sore, its *induration*, and, if obtainable, the time of inoculation, are diagnostic data. The sore is single, and there is hard, nodular, painless swelling of the neighboring lymphatics. A single ulcer of ulcerative stomatitis may in some degree simulate the appear-

FIG. 388.



Chancre of the lip.

ance of a very small chancre. It may exhibit *slight* induration, but its irregular form, situation, painfulness, and the usual absence of lymphatic involvement, together with its prompt disappearance after sterilizing the mouth and cauterizing the ulcer, will differentiate the two sores. If the chancre be upon the tip or sides of the tongue, where it is subjected to irritation, it may become very large and bear a close resemblance to epithelioma of that organ.

It is a wise precaution to view all sores about the mouth as possibly infectious. All errors of diagnosis in this direction will be more than compensated for by the assurance of non-transference of infection.

SECONDARY SYPHILIS OF THE MOUTH.—The secondary manifestations of syphilis are observed in and about the mouth, no matter what the location of the primary lesion may have been; they are the result of a general, not a local, infection.

Secondary affections of the mucous tissues appear in from four to twelve weeks after the appearance of the primary lesion. Sore throat, due to inflammation of the mucous membrane of the pharynx and parts about, is almost constant; together with syphilitic hoarseness, due to the extension of the affection to the mucous membrane of the larynx.

The appearance of copper-colored areas upon some portion of the mucous membrane, on the tonsil, pharynx, soft palate, lips, or buccolabial surface, precedes the loss of epithelium over these surfaces, which soon occurs, forming the most virulently contagious lesion of syphilis, the mucous patch. The patches become covered with a grayish-white pasty

covering, resembling the ulcerations of non-specific stomatitis. So close is the resemblance that a differentiation can only be made at times by additional evidences of secondary syphilis. Single patches may coalesce, forming large irregular areas covered by a grayish-white pellicle. These patches are rarely painful. Ulcerations having ragged, irregular outlines may appear at the sites of the original patches or in other situations, and exhibit a tendency to spread.

The diagnosis of the condition is determined by a discovery of other lesions of secondary syphilis; skin-eruptions, falling out of the hair (alopecia), and the areas of copper-colored eruption upon the mucous membrane of the pharynx and soft palate.

Hugenschmidt[1] has observed among syphilitics, who presented no local lesions, the frequent nocturnal occurrence of indefinitely located dental pains, spreading to the palatal region.

Tertiary Syphilis of the Mouth.—The syphilides of the secondary stage arise in, and are confined to, the mucous and dermal structures; those of the tertiary stage arise in the deep connective tissues, and are frequently associated with periosteum.

Tertiary lesions, as seen by the dentist, are usually in the form of ulcers of, first, the soft or hard palate, and of the tongue or lips. In the earlier stages hard nodular formations may be noted as antecedents to the ulcerations. Chronic periostitis of the palatal processes may occur, leading to the formation of localized thickenings. In other cases, in the soft palate, upon the tongue, or in the hard palate, localized swellings may occur; the overlying mucous membrane breaks, establishing an ulcer, which may perforate the soft palate, and destroy a portion of the palatal process, or form large ulcers on the tongue. These lesions appear in from two to five years after the secondary manifestations.

Although there is much doubt as to the degree of infectiveness of these tertiary lesions, precautions as to sterilization should be taken as with the primary and secondary lesions. A defined, ragged ulcer occupying the hard or soft palate, which has persisted for a long time, should always be viewed with suspicion, and a search be made for other evidences of syphilis.

These ulcerations appearing upon the side of the tongue may closely simulate epithelioma of that organ. The confusion is increased if, in consequence of the presence of jagged teeth, a continuous irritation is excited. Moreover, leukoplakia of the cheeks, a diagnostic sign of incipient epithelioma, frequently accompanies tertiary syphilis. In some cases an absolute diagnosis is only made by noting the disappearance of the local lesion following the administration of iodids, the specific treatment of tertiary syphilis.

[1] *Dental Cosmos*, 1892.

The existence of tertiary syphilis is of great clinical importance to the dentist in that a condition of lessened resistance of tissues is established, and disease-processes which in the healthy person are comparatively circumscribed, in the syphilitic run a riotous course. A septic pericementitis by extension may involve a wide area of periosteum, leading to extensive maxillary necrosis.

Tuberculosis of the Mouth.—The bacillus of tuberculosis, under favorable conditions, develops in the tissues of the mouth, producing its characteristic lesions. Finding a suitable soil, such as is furnished by the heredity which predisposes to phthisis pulmonalis, the bacillus may find entrance to the deeper tissues from the mucous membrane of the mouth and excite tuberculosis in the deep structures, the bone, etc. What part is played by local oral and dental lesions in tuberculosis of distant parts, by establishing pathways for the entrance of the bacilli into the circulation, is at present conjectural, but that such infections occur is very probable.

Actinomycosis.—The condition produced by the development of the ray-fungus, the actinomycosis, in the lower jaw and cervical regions of cattle and swine—lump-jaw—is not unknown in human beings.

Miller[1] gives 203 cases reported in German medical literature between 1886 and 1891. In at least 120 of these cases the point of entrance of the fungus was found to be in the region of the mouth or throat. Actinomycosis-threads have been repeatedly found in the saliva and in carious teeth, and notably in the tonsils. Whether the path of entrance to deeper structures is ever through carious teeth is undetermined, but certainly lesions or wounds about the mouth furnish an entrance.

General Septic Diseases of Dental Origin.

The effect of the existence of dental diseases upon the body at large, particularly as regards secondary infection, is a matter increasing in importance as the possibilities of their connection are made out. At present, the organisms of greatest demonstrable pathological interest are the pyogenic cocci. The almost constant presence of these organisms in the mouth, carried thence into the pharynx, posterior nares, larynx, and lungs, furnishes the reason for the pyogenic inflammations which occur in these organs. The diplococcus of pneumonia, a frequent organism, but waits a favorable opportunity to establish high inflammations and fibrinous exudations in the lungs, and possibly in other structures.

The most important clinical associations of dental with general infections, are diseases of the pericementum. The pulps of teeth, having no lymphatics, do not appear to take up and transmit the

[1] *Dental Cosmos*, 1891.

products of the action of septic organisms; but while the evidences of such absorption, involvement of the neighboring lymphatics, are not present, it must be remembered that the veins may transmit the poison, and, in addition, may perhaps convey organisms from a diseased but still vital pulp to distant parts. When, however, the pulp is dead and the pericementum is invaded, there is no doubt of general infection from this local source. More or less septic intoxication is a common attendant upon severe septic apical pericementitis, and septicæmia accompanied by inflammation of the neighboring lymphatic glands is of sufficient frequency to emphasize the need of the vigorous antiseptic treatment recommended in all of these cases.

Pyæmia is far more uncommon.[1] Pyogenic organisms, gaining access to the blood-current from the local source of infection, establish suppuration in distant parts; in other parts of the bone, or in other bones (osteomyelitis), in the lungs, meninges and substance of the brain. One case[2] has been reported where abscess of a toe, ear, and forearm ceased, and recovery took place after treatment and filling of septic root-canals. Several cases are tabulated by the same author in which extensive necrosis and death resulted from pyæmic infection from septic pericementitis. Some of these cases recorded were associated with acute, some with chronic septic pericementitis.

In addition to the usual pyogenic cocci, Miller has isolated several forms of cocci, bacilli, and spirilla, forming products, which, if injected into the circulation of animals, cause death from septicæmia in from hours to days. As many of these forms may be brought into relation with deep parts by the anatomical conditions created by pulp-death, the possibilities of many types of infection *via* pulpless teeth are evident.

The possibilities of infections through the conditions established in the several forms of pyorrhœa alveolaris should not be forgotten.

The pockets formed by the soft tissues overhanging lower third molars whose eruption is impeded invite the passage of septic organisms to deep parts. Local pyogenic infections are common in these cases.

Dental Sterilization.

It must ever be borne in mind that the dental operator constantly works in a field of infection, and unless extraordinary precautions be taken every instrument which touches this field—the fingers of the operator, his mirrors, glasses, napkins, rubber-dam, rubber-dam clamps—becomes immediately infected as soon as it is brought in contact with the mouth of the patient. The likelihood of infection varies with the patient and the particular instruments; mouth-mirrors, rubber-dam

[1] Miller, *Dental Cosmos*, 1891. [2] *Ibid.*

clamps, scalers, and all instruments used in the treatment of pulp-canals, are likely to become more promptly and extensively infected than other instruments. Again, the forms of the instruments determine whether or not increased opportunity is given for the retention of infective material. The fingers of the operator may be the medium through which infective material is transferred from one patient to another. Infection may be carried from superficial areas of the mucous membrane of the mouth, from the enamel and the saliva, into deeper structures, where conditions are favorable for the development of sepsis.

The scheme for dental sterilization, therefore, includes the sterilization of the operator, instruments, apparatus, appliances, etc., used in operations, and the sterilization of the field of operation prior to operating.

THE OPERATOR.

Extreme personal cleanliness upon the part of an operator is clearly the first step in asepsis. The best class of dentists are exceedingly neat as regards personal habits: daily bathing, care of the nails and of the skin, and immaculate linen, form as much a part of the day's labor as dental operations *per se*. The virtues of soap and water, wherever they may be applied, are regarded as a very important item in preventing infection.

Linen which has been boiled prior to wearing may be regarded as safely sterile; so that the matter of personal sterilization relates to the hands, particularly to the finger-nails. The space under the nails is a favorable habitat for many organisms, notably the pyogenic cocci, the staphylococcus pyogenes aureus being commonly present in this situation.

It has always been advised that the finger-nails be trimmed short, and be made smooth to avoid mechanical injury to the soft tissues of the patient. Since the advent of aseptic and antiseptic surgery these precautions have an additional significance; nails kept short and smooth may be more readily cleansed than if long and ill-kept. The nails should be cut with a sharp knife-blade so that they nowhere project beyond the tips of the finger. Their mechanical cleansing should be done with smooth instruments, not sharp knife-blades; the latter produce rough surfaces, which furnish spaces for lodgement of bacteria. There is but one effective method of washing beneath the nails; it is that followed by the general surgeon: after dipping the soap in water as hot as can be borne by the hands, all of the finger-nails should be made to scrape the soap until the spaces under the nails are filled with soap. After this, coarse hand-brushes are used to scour every part of the hands with soap and water as hot as can be borne. Special nail-brushes are next used to scrub beneath the nails, driving out piecemeal the soap-masses there. The general surgeon continues the scrubbing until the nails are scrupu-

lously clean. The soap usually used is Castile, or soap made from palm oil, etc.; but antiseptic soaps may be substituted with advantage.

Sterilization of the cleansed hands is insured by immersing them in antiseptic solutions, such as a 1 : 1000 solution of mercuric chlorid. The hands should be sterilized after treating each patient. If the patient dismissed have possessed an unusually septic mouth, or have been a syphilitic, for example, the time for hand-cleansing and sterilization is to be prolonged; if syphilitic, every instrument used is transferred to separate vessels containing antiseptic solutions, and the hands are viewed as highly infected; they are scrubbed with mercuric chlorid solutions to prevent personal infection or the carriage of infection.

STERILIZATION OF APPARATUS.

The scrupulous cleanliness of the operating-chair, whose head-rest should receive frequent changes of boiled linen coverings, metallic parts rubbed, and general covering cleansed; the cleansing, polishing, and sterilizing of cuspidores; the changing of lining coverings upon instrument-tables, etc., are part of the general scheme of sterilization. The floor of the operating-room also requires attention; instead of being covered with carpet, it is preferable to have it made of parquetry material, lacquered hard wood, over which rugs are laid, which may be removed from the room for cleansing, the floor proper being scrubbed.

Glassware, such as tumblers, may be effectually sterilized by boiling. Linen napkins are also sterilized in the same manner. Napkins used about the mouth are certain to become infected, so that their boiling should be prolonged at least fifteen minutes. For many operations it is preferable to substitute strips of muslin for linen napkins, which after being used may be thrown away.

If a hydraulic saliva-ejector be used, the glass mouth-tubes should be changed for each person, a sterilized tube being immediately substituted as soon as a patient leaves the chair. These tubes should receive prolonged boiling before a second use. At the close of each day a large cup should be filled with an antiseptic solution, which is to be drawn through the tubing of the ejector to keep it in a reasonably aseptic condition.

Rubber-dam may be sterilized by boiling water, but it is more safe and cleanly to use a new piece for each patient. The possibilities of infection through this medium are great, particularly in syphilitic cases.

STERILIZING INSTRUMENTS.

The sterilizing of instruments comprises their mechanical cleansing and the use of germicides; steam heat and antiseptic drugs are both employed for this purpose. Steam heat being the most convenient and

certain sterilizing agent, is used wherever it cannot produce injury to instruments.

All instruments should be kept in a highly polished condition, being rubbed with crocus cloth at the end of each day's use. Excavators, explorers, and pulp-canal cleansers should be mechanically freed from visible foreign matter by rubbing their points with a wire brush. A wire brush should be used to cleanse all excavating-burs which have been in use. To sterilize the mechanically cleansed instruments they are boiled in water to which 2 per cent. of sodium carbonate has been added to prevent rusting.

Mouth-mirrors, of which there should be several, require special care. Their edges afford favorable lodging-places for bacteria, and hence they require long boiling. Miller[1] found that the usual antiseptic solutions used cold acted as very imperfect sterilizers, but at the temperature of boiling their efficacy was markedly increased.

Extracting-forceps require careful mechanical cleansing and prolonged boiling after each use, for perhaps more cases of infection, and of many kinds, have resulted from dirty forceps than from all other causes combined.

STERILIZING THE FIELD OF OPERATION.

To insure sterilization of the field of operation antiseptics should be used freely about the mouth prior to operating. The thoroughness of the sterilization will depend in great degree upon the personal habits of the patient. If by the systematic use of the agents and measures described under the prophylaxis of caries, the patient's mouth be kept in a reasonably aseptic condition, sterilization of the oral cavity can be accomplished with sufficient readiness. The choice of antiseptic will depend in great degree upon the state of oral hygiene; in ill-kept mouths, with deposits of foreign materials about and between the teeth, on the gums and tongue, much more active and penetrating germicides will be required than if the parts are clean. The presence of putrefactive decomposition in the mouth, made evident by ill odors, amid which that of hydrogen sulfid may be detected, needs for its treatment the immediate and free use of preparations from which nascent chlorin or nascent oxygen may be disengaged. No operation or even examination should be begun in such cases before a claret-colored solution of potassium permanganate, or a strong solution of hypochlorites (meditrina diluted), has been freely used by the patient. Many operators keep a stock of inexpensive tooth-brushes for such cases, which are thrown away after the patient has used them, who is directed to scrub the teeth well with brush and the antiseptic solution.

[1] *Dental Cosmos*, 1891.

The routine practice of scaling and polishing the teeth and prescribing an antiseptic mouth-wash prior to the commencement of a series of sittings, is to be highly commended. To sterilize a comparatively clean mouth sufficiently to begin dental operations, hydrogen dioxid may be used; it should be held in the mouth and pumped about by the movements of the lips and cheeks for a minute or longer.

If ulcerations or inflammatory conditions exist, the sterilization is to be prolonged, using such agents as meditrina. If a suspicion of syphilis exist, not only should the mouth be freely washed with strong antiseptics, but special instruments should be used, preferably an old set, kept sterilized and used only in special cases. If the hands of the operator have abrasions or irritated spots, they should be covered with collodion, or, better, the examination should be referred to one whose skin-surface is unbroken.

SECTION VII.

DENTAL PHARMACOLOGY AND DENTAL MATERIA MEDICA.

DENTAL PHARMACOLOGY.

A DENTAL materia medica includes a description of the agencies which are employed in the treatment of oral diseases; dental pharmacology considers the mode of action and composition of these substances and agencies. The intelligent use of drugs, as of any therapeutic resource, is based upon a primary knowledge of their chemical and physical properties, together with a familiarity with the exact nature of the altered physiology the practitioner is called upon to remedy. A rational therapeusis is founded upon the utilization of the specific properties of remedial agents to combat morbid conditions whose character has been clearly determined.

If generalizations be made of the several disease-states described in the body of this volume, it will be seen that the dental practitioner meets with three great classes of conditions requiring treatment:

First, a large majority of diseases of the teeth and associated parts are bacterial in origin—are due to septic influences; hence the first group of agents of dental practice are those used to combat septic conditions, or antiseptics.

The second class of conditions demanding relief are those in which pain is a prominent symptom; hence the second class of agents to be considered are those used for the relief of pain—anæsthetics or analgesics.

The third group of conditions, viewed as a class, comprises those characterized by a relaxation of soft tissues and a passive dilatation of their bloodvessels; hence the third group of dental remedies is that of the astringents, or substances which have the power to bring about contraction of relaxed parts.

Nearly all of the agents which have been found to possess distinct therapeutic value in dentistry may be placed under one of these three heads, although an agent of one group may possess properties of all three classes.

Some few of the drugs and agents employed in dental therapeusis cannot be properly classified under any of the three heads named, but

these, as well as all other therapeutic agents, may be included under two heads—stimulants and sedatives; that is, all agents used in general or special medicine are employed either to exalt or to depress some one, or more, vital functions of some organ or tissues of the body.

Antiseptics.

Antiseptics are agents which prevent the action of pathogenic organisms or of their products upon the living body. Members of this great group differ in their chemical properties and mode of action. Under the head of antiseptics are grouped sub-classes of remedies, named in accordance with their mode of action against septic influences. They may act by destroying the vitality of the infective organisms; a substance or agency having this power is termed a germicide. They may act by chemically destroying the poisons formed by bacteria without necessarily killing the organisms. Conditions may be established in the cells of the body which inhibit the growth of organisms. Any agent which has the power to remedy an existing infection is called a disinfectant. It is seen, therefore, that a distinction may be drawn between an antiseptic, a germicide, and a disinfectant. A germicide is both antiseptic and disinfectant, but antiseptics and disinfectants are not necessarily germicides. Again, an antiseptic acts to prevent as well as remedy infection; a disinfectant implies an existing infection. Under this head should be grouped deodorants, agents that have the power of destroying objectionable odors which arise during putrefactive processes, and from other sources. A true deodorant operates by chemically destroying the malodorous substance; for example, chlorin vapor acts as a deodorant by abstracting the hydrogen from such a substance as hydrogen sulfid, destroying its chemical identity and therefore the original odor:

$$H_2S + Cl_2 = 2HCl + S.$$

Bromin, iodin, and nascent oxygen act after the same manner. Most of the deodorants, and all of those named, are active antiseptics, many of them germicides, destroying not alone the organisms, but also their waste-products. Agents such as carbolic acid and the cresols are not true deodorants, although they destroy the organisms which are the primary cause of the offensive odors.

Nearly all of the antiseptics employed in the treatment of dental diseases may be grouped under eight heads:

First.	Salts of metals . . .	{	Zinc chlorid; mercuric chlorid; silver nitrate, citrate, and lactate; copper sulfate.

Second.	Alcohols and their derivatives	Ethyl alcohol; methyl alcohol, and its derivative formaldehyd; phenyl alcohol, including all allied substances, such as creasote, the cresols, creolin, and lysol.
Third.	The halogen group and their compounds . .	Iodin and its preparations, including iodoform, nosophen, aristol, iodol, antinosin; solutions from which nascent chlorin may be evolved—hypochlorites; bromin is not used as a dental disinfectant.
Fourth.	Mineral acids	Sulfurous, sulfuric, hydrochloric, chromic, and boric acids; solutions of hyposulfites, which act by virtue of their sulfite radical, may be included.
	Organic acids	Trichloracetic, lactic, and benzoic.
Fifth.	The caustic alkalies .	Metallic sodium and potassium in alloy (kalium-natrium); the hydrates and carbonates of sodium and potassium; sodium dioxid; aq. ammoniæ fort.
Sixth.	Solutions from which nascent oxygen is evolved.	Hydrogen dioxid in watery and ethereal solutions from 3 per cent. to 25 per cent. strength; solutions of sodium dioxid; potassium permanganate.
Seventh.	The essential oils . .	Cajuput; cassia; cinnamon; cloves (and eugenol); eucalyptus; gaultheria; myrtol; and thyme.
Eighth.	Physical agencies . .	Heat, dry and moist.

Each of these groups exhibits distinct modes of germicidal action, in accordance with their chemical properties.

The varied conditions under which bacteria are found will modify the choice and application of antiseptics and germicides. To act as a direct

and prompt germicide the agent must be brought into intimate contact with the organisms. There are physical obstacles, varying under many conditions, which antagonize the desired end. Many forms of bacteria are enclosed in a resistant coating or cell-wall; in addition to this, several forms unite into large colonies, zoöglea, through the medium of an albuminous excretion. Even under favorable conditions the germicide must penetrate the mass to effect sterilization. In addition to this, the offending organisms may lie deep in a mass composed of fatty and albuminous matter, so that the question of the diffusibility of the antiseptic is an important one.

"An antiseptic agent must be of sufficient strength to destroy the disturbing bacteria.[1]

"If bacteria develop so that the germicide can surround or easily penetrate them, they are more readily killed than when they develop in mass or clump-forms, and are surrounded by protective matter, as we find them in dental caries, root-canals, etc. In this state a stronger solution of the disinfectant is generally necessary, and a longer time will be required for the agent to penetrate the mass and act on the micro-organisms.

"An agent should be employed that will not limit its own action and thus fail to reach all noxious germs. For instance, where the bacteria are imbedded in albuminous material, oxidizing agents, as hydrogen dioxid, sodium dioxid, potassium salts, etc., will disintegrate and permeate the mass, while such agents as bichlorid of mercury and silver nitrate are apt to combine with the albuminous material and coagulate it, and thus limit or delay their own action. In this condition, the bichlorid may act as an antiseptic and restrain growth until the mercury is partly removed by the action of ammonium or hydrogen sulfid; then the unkilled germs, or spores, will again develop if the nutrient material be favorable for their growth. Diluted solutions of such an agent, however, are not apt to cause so dense a coagulum as to prevent permeation throughout the mass; therefore a 1 : 1000 or 1 : 2000 solution of bichlorid of mecury is considered most desirable. The addition of 5 parts of tartaric acid to 1000 of the solution assists materially in preventing coagulation by bichlorid solutions (Laplace).

"Germicidal agents that are incompatible should not be used together or one immediately after the other. Some agents become inert through chemical reaction when brought in contact with putrefying matter. Bichlorid of mercury, especially in dilute solutions, is rapidly decomposed by sulfids, alkalies, ammonium salts, and even organic matter.

[1] These conditions and their connection have been well summarized by L. P. Bethel, in "Prize Essay" quoted.

Therefore its use as a dressing for root-canals containing putrescing material is of doubtful value.

"Very volatile antiseptics should not be used where a continued action is desired, as in root-canal treatment.

"Fresh solutions should be used. Many preparations deteriorate with age, exposure, etc.

"Again, the potency of germicides and antiseptics is increased by combining medicaments. For instance, the germicidal power of bichlorid of mercury is increased by the addition of hydrogen dioxid, benzoic acid, etc., and carbolic acid mixed with sulfuric acid increases its efficacy. When these and other disinfecting agents are combined with alcohol, glycerin, or oil, however, the germicidal power is greatly lessened.

"Laboratory experiments show that bichlorid of mercury as strong as 1 : 250 solution in absolute alcohol, or 1 : 50 in glycerin, does not destroy well-exposed anthrax spores in two days' time; but a 1 : 1000 solution in alcohol plus 15 per cent. of water, or 1 : 500 in glycerin plus 50 per cent. of water, will destroy them in twenty-four hours.

"Oil has about the same effect as glycerin. The results with carbolic acid, 1 : 10 solution, are similar. The advantage of using aqueous solutions when possible, is obvious.

"Laboratory experiments show also that a warm or hot germicidal solution is more effective than a cold one. A 5 per cent. solution of carbolic acid at 70° F. requires one month to kill anthrax spores. Heated to 104° F. it kills them in about six hours; at 135° F., in one hour and a quarter; at 180° F., in fifteen minutes. A 1 : 1000 bichlorid of mercury solution heated to 115° F. is equal in disinfecting power to a 1 : 500 solution used cold. Experiments made with various bacteria to determine why warm or hot solutions make this difference seem to indicate that not only is the vitality of the bacteria lowered, but the bacterial cell is actually enlarged or swollen, probably thus increasing the absorptive surface.

"The thermal death-points, with moist heat, of some of the pus micro-organisms, are—bacillus pyocyaneus, 132° F.; staphylococcus pyogenes aureus, 136° F.; citreus, 143° F.; albus, 143° F.; cereus flavus, 134° F.; cereus albus, 134° F.

"This points out the advantage of using hot injections of water or disinfectant solutions in pyorrhœa alveolaris, pulpless teeth, antral and other diseases.

"For germicides to act most effectively the field of operation should be as thoroughly prepared as possible previous to their application. For example, in root-canal treatment some instrumentation should precede the application of the dressing, to break up the compact masses and permit the germicide to penetrate to the organisms.

"Grease should be removed by aqua ammoniæ, equal parts of ether and alcohol, or chloroform; and all other foreign substances, which might hinder the action of germicides used, should be removed."

SALTS OF METALS AS ANTISEPTICS.

The salts of metals are used in watery solutions as antiseptics—mercuric chlorid usually in 1 : 1000 solution; zinc chlorid, 1 : 100; silver nitrate, 1 : 100; copper sulfate, 1 : 100. These are about the strengths in which these agents are found to act as efficient germicides; but in the interiors of teeth, where the question of general poisoning rarely is a factor, and local poisoning only a remote possibility, these strengths may be greatly increased.

Metallic salts act as germicides by chemically destroying protoplasm. When a metallic salt, in sufficient solution-strength, is brought in contact with albuminous material an obscure chemical change occurs, the metal entering into combination with the albumin and forming a substance which, for want of a better name, is called an albuminate of that metal, an opaque body replacing the transparent gelatinous albumin. For example, when mercuric chlorid is brought in contact with albuminous material, such as serous effusions, the serum of the blood, or living cells, animal or bacterial, the identity of the albuminous matter is destroyed. If a percentage of an organic acid, say tartaric (Laplace), be added to the mercuric solution before it is brought in contact with the albuminous matter, the above reaction does not appear to occur. The albuminate of mercury thus formed is decomposed under suitable conditions. If ammonium sulfid be added to an albuminate of mercury, mercuric sulfid is formed and the albumin is set free. This reaction is of importance as regards the use of mercuric chlorid as a disinfectant where sulfur compounds are present, as in putrefactive fermentation, when hydrogen sulfid is formed, a similar reaction may occur, and antiseptic effects be neutralized. During the putrefactive decomposition of albumin other substances than H_2S, capable of decomposing mercuric chlorid, are formed.

Mercuric chlorid inhibits the vital activity of living cells, even in very dilute solutions. A solution of 1 : 20,000 checks the movements of spermatozoa; in one of 1 : 2000 it acts as a germicide to most micro-organisms, although several forms which have a resistive cell-wall delay the germicidal action. The spores of some species of bacilli, notably those of the bacillus anthracis, are markedly resistant. The precipitate of mercury albuminate which occurs interferes with the germicidal action of the solution; the addition of tartaric acid to the mercury solution aids its germicidal property by preventing the formation of mercury albuminate.

While all of the metallic salts named form with solutions of albumin coagula, or albuminates of the metals, they differ in the rapidity of the chemical change and in the character of the coagulum.[1] "Capillary tubes containing a mixture of albumin and 20 per cent. of glycerin, the glycerin being added to prevent the drying of the albumin. One end of the tube was sealed, and saturated solutions of antiseptics applied at the other end for a period of ten days. Contrary to the belief generally held, silver nitrate was found to effect deep coagulation, the coagulum becoming dark in color; zinc chlorid produced complete white coagula."

These facts are of great importance, since they prove that the agents named, instead of being self-limited in action, may by prolonged contact affect deeper structures.

Being a chemical reaction, there is, of course, as pointed out by Kirk,[2] a quantitative relationship between the substance acting and the substance acted upon.

The presence of a coagulum of albumin between parts to be acted upon and the germicide must delay and interfere with the free diffusion of the germicide, as taught by Harlan; but if the germicide be present in sufficient amount, it will diffuse through the coagulum, as demonstrated by the experiments of Kirk and Truman.

Secondary changes have been noted in the coagulum with mercury. The coagulum of zinc chlorid is very resistant and appears to possess permanent antiseptic properties. The coagulum of silver nitrate undergoes chemical changes: the albuminate of silver is reduced to an oxid, and, if brought in contact with acids, forms corresponding salts of silver. For example, in contact with lactic acid silver lactate, a persistent antiseptic, is formed.

These agents, used in sufficient strength, form coagula with the protoplasm of cells, coagulating intercellular albuminous material and forming an eschar—*i. e.*, they are caustics.

ALCOHOLS AND THEIR DERIVATIVES AS ANTISEPTICS.

The alcohols and their derivatives also act as germicides by effecting coagulation of albuminous bodies. They differ in the rapidity of their action and in their germicidal strength. The cresols, trikresol having the greatest antiseptic power, carbolic acid next, and ethylic alcohol least. Formaldehyd in watery solution is the most promptly acting and penetrating agent of this group.

HALOGEN DERIVATIVES AS ANTISEPTICS.

The halogen group has its activity represented by the chemical affinities of iodin and chlorin. These agents abstract hydrogen from organic com-

[1] Truman, *Proc. Academy of Stomatology*, 1894. [2] *Dental Cosmos*, 1893.

pounds, and thus destroy their identity. While it is certain that they act as germicides, destroying the vitality of protoplasm, their principal field of usefulness is in rendering innocuous the products of bacterial decomposition. The iodin derivatives named under this head are supposed to set iodin free when brought in contact with infected tissue, the iodin acting as a persistent disinfectant. Their activity is gauged, therefore, by the amount of iodin set free—*i. e.*, by the amount present and the looseness of its combination.

ACIDS AS ANTISEPTICS.

The mineral acids effect the chemical destruction of animal substances, inorganic and organic, with which they are brought in contact. They differ in their activity in this direction and in the depth of their action. For example, sulfuric acid quickly forms a black eschar upon tissue, chromic acid penetrates deeply into tissues to which it is applied. The milder acids, such as boric acid, appear to have no escharotic action.

The strong mineral acids abstract the potassium, sodium, and calcium elements from tissues with which they are brought in contact, in addition to destroying albumin. The organic acids have a similar action.

Solutions of hyposulfites act by virtue of the sulfite radical, SO_2, which abstracts the elements of water from organic matter, forming sulfurous acid, H_2SO_3. The sulfite radical formed by burning sulfur acts in the same manner.

CAUSTIC ALKALIES AS ANTISEPTICS.

The caustic alkalies chemically destroy albuminous matters with which they are brought in contact. They combine with the fatty acids produced during putrefactive decomposition of tissues, and form soaps. The hydrates of sodium and potassium are the most active. These substances are formed when the alloy of sodium and potassium is brought in contact with organic matter. The alloy abstracts hydroxyl, HO, from organic matter, and produces secondary decompositions by the hydroxids of the metals which are formed. Sodium dioxid (Na_2O_2), in contact with organic matter, sets free its loosely held oxygen atom, and the sodium oxid left quickly abstracts water from the organic matter, forming sodium hydroxid:

$$Na_2O_2 + \text{organic matter} = N_2O + O +.$$
$$Na_2O + H_2O = 2NaHO,$$

which acts as above described.

Aq. ammoniæ fort. acts by virtue of its affinity for water and fatty acids.

THE ESSENTIAL OILS AS ANTISEPTICS.

The essential oils possess in varying degree the property of destroying bacteria; while it is probable that they effect a change in albuminous substances akin to coagulation, it has not yet been clearly demonstrated. Certainly they act as poisons when brought in contact with protoplasmic substances. Their germicidal power varies, that of the oils of thyme and cinnamon being greatest; that of cloves (eugenol also) is less marked. Placed in the roots of pulpless teeth, they diffuse very gradually through the contents of the dentinal tubules. They may cause distinct staining of the tooth, a proof of their diffusion.

PHYSICAL AGENCIES AS ANTISEPTICS.

Of the physical agencies, the only available germicide is heat. It produces coagulation of albuminous matter, and is thus antiseptic. The degrees and conditions of heat necessary to complete germicidal action vary as to the mode of applying the heat and also upon the nature of the organisms present. Moist heat is a much more effective germicide than is dry heat.

A degree of heat fatal to any mature organism may not destroy the vitality of its spores. The spores of some bacilli resist a temperature of boiling water for several hours. Most organisms, however, are promptly killed by water at a temperature of 212° F.

ANÆSTHETICS.

An anæsthetic is any agent which prevents the perception of pain. A condition of insensibility to pain, or tactile sensitivity, may be induced in three ways: first, by paralyzing or destroying the terminals of sensory nerves through which impressions of pain are conducted—that is, by abolishing reception; secondly, by interfering with or preventing the transmission of such impressions after their reception; thirdly, by so acting upon the perceptive centre of the brain that its function is held in abeyance—*i. e.*, by abolishing perception.

Agents which have the power of lessening the consciousness of pain are grouped under the heads of anodynes, analgesics, and obtundents. The term anæsthesia, as originally used, indicated a condition of insensibility attended by loss of consciousness; hence the term anæsthetic is usually employed to designate such substances as derivatives of ethane, notably ethylic oxid or ether; derivatives of methane, such as methyl trichlorid or chloroform; and nitrous oxid gas.

An anodyne is an agent acting upon the pain-perceptive centre of the brain in such a manner that perception of pain is benumbed or lost; the term refers to abolishing existing pain, as by the action of opium derivatives.

An analgesic is an agent which either prevents or subdues pain, and may act upon any portion of the sensory tract.

Under the head of obtundents are included those agents which are applied locally to benumb the terminals of sensory nerves.

Hypnotics act as analgesics by inducing sleep, during which common sensation is in abeyance.

The typical general anæsthetics are those named above; chloroform as the representative methane derivative; ether, the ethane derivative; and nitrous oxid, the asphyxial anæsthetic.

Chloroform and ether act as general anæsthetics by abolishing the functions of the centres of consciousness in the cerebrum, and that of pain-perception. "They probably enter into loose combination with the protoplasm of the cortex of the brain, producing a temporary fixation, and interfere with the process of oxidation and reduction upon which the functions of these cells depend."[1]

The effects of these agents are progressive: the higher cerebral functions are first abolished; next the special sense-perceptions; then reflex activities; until, finally, the centres which preside over the vital functions of respiration and circulation are involved, and if administration be carried beyond this point, death results.

In general terms, these anæsthetic agents and their kindred may be said to be dangerous to life in proportion to their vapor-density. The greater their vapor-density, the longer they remain in the body.

Nitrous oxid, under the usual conditions of administration, induces general anæsthesia by the exclusion of oxygen from the lungs and the accumulation of carbon dioxid in the blood; in addition, it has a specific anæsthetic action, as unconsciousness and anæsthesia are induced when the nitrous oxid administered is mixed with oxygen. Nitrous oxid is to be regarded as the only entirely safe general anæsthetic.

The number of fatalities attending the use of chloroform, the list being particularly large in connection with its administration for tooth-extraction, is sufficiently extensive to absolutely contraindicate its use in that connection. The danger is twofold: first, the erect posture, which favors and appears to precipitate syncope; secondly, the partial anæsthesia. More deaths are recorded as occurring in connection with partial than with full chloroform-narcosis; probably under partial anæsthesia the reflexes are not entirely lost, and a condition of profound shock ensues upon performing minor operations.[2] Death under chloroform in some cases is due to paralysis of respiration; in others, of the circulation. In many of the cases, when respiration ceases and the heart continues

[1] Lauder Brunton, Croonian Lectures, 1888.

[2] See Brunton, *Pharmacology and Therapeutics.*

to beat, life may be preserved if artificial respiration be maintained long enough for the body to rid itself of the chloroform-vapor.

Representative anodynes, agents benumbing the pain-perception centre of the brain, are morphia and several of the coal-tar derivatives. These agents probably act in a manner similar to that noted in connection with the general anæsthetics, by forming loose combinations with the protoplasm of nerve-cells. Many of them affect the nervous paths, and when locally applied to sensory nerve-terminals reduce their function, so that they may act as analgesics under all three heads named.

The coal-tar derivatives are chemically the analogues of the vegetable alkaloids; that is, they are substitution-products of ammonia. By replacement of the several hydrogen atoms of the ammonia base predetermined properties may be conferred upon compounds. Beginning with replacement by a phenyl radical and adding, for example, the anæsthetic basis methyl, analgesic substances are produced. For example,[1] NH_3, ammonia, may have two of its hydrogen atoms replaced by other radicals:

$$N\begin{cases} C_6H_5 \\ H \\ H \end{cases} = \text{anilin}$$

$$N\begin{cases} C_6H_5 \\ H \\ COCH_3 \end{cases} = \text{phényl acetamide or acetanilid, an analgesic agent.}$$

The remaining hydrogen atom may be replaced by the anæsthetic radical CH_3, methyl, increasing the analgesic power of the compound:

$$N\begin{cases} C_6H_5 \\ COCH_3 \\ CH_3 \end{cases} \text{forming methyl acetanilid, or exalgin.}$$

Displacements and replacements in such comparatively simple bodies may be made, changes of physiological properties following upon changes of chemical composition.

These agents have the power of paralyzing the paths of pain-conduction.

Equally instructive observations may be noted in connection with local anæsthetics, those agents which possess the power of benumbing the terminals of sensory nerves. Excluding such agents as volatile hydrocarbons, rhigolene, ethyl and methyl chlorid, in which specific paralyzing action is masked by the intense cold produced by their application, and inducing analgesia, it will be seen that the best-known

[1] Brunton, Croonian Lectures, 1888.

and most active local anæsthetics are all related to one another in chemical composition. These agents are atropia, homatropin, tropacocain, cocain, and eucain.

Atropia is a compound of tropic acid with tropein, a tropate of tropein; either of these substances alone possesses no analgesic power, but the combination is slightly analgesic. If, however, tropein be combined with benzoic, instead of tropic acid, benzoyl tropein is formed (homatropin), which possesses marked anæsthetic properties.

The next member, tropacocain (benzoyl pseudo-tropein), bears a chemical relationship to the former, and has additional anæsthetic power.

Cocain is chemically benzoyl methyl ecgonin; when boiled it is split up into methylic alcohol, benzoic acid, and ecgonin. The anæsthetic properties reside in the benzoyl methyl, and are much more marked than in compounds in which the methyl radical is absent.

In eucain, the next member, benzoyl and methyl are both present, the latter multiplied and in several combinations, so that the anæsthetic properties are greater than those of cocain.

If ethyl instead of methyl be combined with benzoyl ecgonin, an anæsthetic, cocethylen, is formed.

The benzoyl derivatives of other substances, such as morphia, have anæsthetic properties.

The induction of analgesia by some local anæsthetics is preceded by a stage of irritation.

The phénols are all local anæsthetics, but many of them combine actively with the albuminous portions of tissues and act also as caustics.

The benzoyl derivatives mentioned act not only as paralyzants of nerve-terminals, but, if applied or injected about the trunk of a nerve, induced analgesia in the distribution of that nerve. They paralyze nerve-centres to which they are applied.

The essential oils possess the power of paralyzing sensory nerve-terminals, inducing, first, marked irritation; this effect is absent when they are applied to the dental pulp, a tissue in which tactile sensitivity is normally absent.

Astringents.

Astringents are chemical agents which, when applied to swollen vital tissues, cause their contraction without their destruction. Many of them, however, if used in sufficient strength, immediately destroy the vitality of tissues to which they are applied; for example, zinc chlorid in saturated solution is a powerful caustic; in 10 per cent. solution is an active astringent; and in 1 per cent. solution acts as a stimulant. The reason for this is readily seen from a study of its mode of action.

Astringents are divided into vegetable and mineral; the vegetable

astringents owe their property to the tannic acid contained in them. The mineral astringents are mainly salts of iron, copper, lead, and zinc—acetates, sulfates, and chlorids. Astringents, with but few exceptions, act by causing more or less coagulation of albuminous fluids and a shrinkage of the tissues in which they are contained, and, with the exception of subacetate of lead and nitrate of silver, do not cause contraction of bloodvessels; tannic and gallic acids cause dilatation of vessels.[1] It will be seen, therefore, that, with the exception of silver nitrate and lead acetate, all astringents are positively contraindicated in active inflammation. Their sphere of usefulness is limited to states of venous congestion with effusion; for example, in such conditions as chronically tumid gums, the use of an active vegetable astringent will cause constringing of the swollen tissues and dilatation of the arteries, with an increased flow of blood; the weakened veins will receive support, and stagnation be relieved.

These substances differ in effect in proportion to their affinity for albumin, and the strength in which they should be used will depend upon this affinity; for example, zinc chlorid or silver nitrate used in strength greater than 20 per cent. will combine with protoplasm and cause tissue-death.

In combining two or more remedies regard should be paid to the chemical reactions which may occur between them and alter their nature; drugs which, mixed together, produce undesirable combinations are termed incompatibles. It may easily happen in the practice of dentistry that such mixtures may be made and produce ill-results; for example, if sulfuric acid be used in connection with steel instruments in dental canals, and preparations containing tannic acid be afterward applied, a black tannate of iron forms, staining the dentin; or, again, if attempts be made to bleach discolored dentin with nascent chlorin in teeth containing gold fillings, auric chlorid may form and cause permanent staining. The following list from Hare[2] is a useful summary of common incompatibles:

First. An acid should never be combined with an alkali.

Second. An acid should not be added in any quantity to a tincture.

Third. Alkalies should not be combined with the alkaloids.

Fourth. Potassium chlorate should not be ordered to be rubbed up with tannic acid or any other organic substance capable of oxidation, as it will explode. Permanganate of potassium is subject to the same rule.

Sixth. Iron is incompatible with tannic acid, as it forms a tannate of iron, or ink. As all the vegetable astringents contain tannic acid, none of them should be used with iron, except chiretta and calumba.

Seventh. Tannic acid should never be added to solutions of alkaloids.

[1] Brunton, *Pharmacology and Therapeutics.* [2] *Practical Therapeutics.*

Eighth. Alcoholic solutions of camphor and similar resinous substances are incompatible with water.

Ninth. Fluid extracts are incompatible with water, as the addition of water will cause a precipitate.

Tenth. All salts not acid, but alkaline in reaction, are decomposed by acids.

Eleventh. All salts which are acid are decomposed by alkalies.

Twelfth. All vegetable acid salts are altered by mineral acids and are decomposed by alkalies.

Thirteenth. Iodin and iodids should not be given with alkaloids.

Fourteenth. Corrosive sublimate, the salts of lead, iodid of potassium, and nitrate of silver should always be prescribed alone.

Fifteenth. Cocain and borax when added together form an insoluble borate of cocain. Boric acid and cocain do not form this substance.

Electricity in Dental Therapeutics.

Electricity is utilized in dental therapeusis for its physical, chemical, and physiological effects. For physical and chemical effects the constant or galvanic current alone is employed; for physiological effects both interrupted (faradic) and constant (galvanic) currents are applied; the former rarely, the latter frequently.

Physical Effects.—The physical properties utilized are the correlation of electric currents into light and heat. Small lamps operated at an electric pressure of 8 to 10 volts, placed in proper relation to reflecting-mirrors, are used to transilluminate teeth, to determine the vitality of their pulps. A somewhat less voltage is used in connection with an appropriate hand-piece and a loop of fine platinum wire to generate a high degree of heat in the latter—an electrocautery. This is used to destroy the peripheral portions of the dentinal processes in cases of excessive hypersensitivity of dentin.

"If a long copper point be placed in a pulp-canal, and the electrocautery be brought in contact with its end, sufficient heat is transmitted through the copper to dry the walls of the canal. If the canal be filled with an antiseptic oil prior to inserting the copper point, the oil may be vaporized and driven into the tubules. If wax or paraffin be placed in the canal, the copper point set in position, and heat applied, the substance will be melted and will run into all the interstices of the canal. The metallic point is permitted to remain as the central canal-filling."[1]

Physiological Effects.—An induced interrupted current was used by Bonwill as early as 1859 to paralyze the reaction of the nerves about the root of a tooth, so that the latter could be extracted pain-

[1] Gramm, *Dental Cosmos*, vol. 35.

lessly. The same device has been used to allay the irritability of the dentinal processes and permit painless cutting of dentin.

Rapidly interrupted currents have been applied to relieve the symptoms of pulpitis and acute pericementitis.

A moistened electrode applied to the back of the neck, the other pole upon the cheek, will tend to cause contraction of the blood-vessels.

They appear to reduce also the pain referred to the seat of disease. The effects, however, are much less marked than when a galvanic current is employed. The latter is useful in those cases of trifacial neuralgia in which there is an irregular contraction of arteries. The galvanic current, 30–50 elements, will bring about a uniform contraction of the vessels. This measure is merely palliative, not curative, so that in cases of reflex neuralgia of dental origin it precedes and follows removal of the cause.

Chemical Effects.—The electrolytic power of the galvanic current is utilized in dental therapeutics. If a current such as is obtainable from the cataphoretic apparatus, with a maximum voltage of 35 to 40, be passed through vital tissues,[1] the electro-positive elements of the tissues, potassium, sodium, calcium, and hydrogen, appear at the negative pole, and acids, chlorin, and oxygen at the positive pole. "It follows that if the positive electrode be composed of metal, it will be corroded by the action of the chlorin and acids, and the negative will remain unacted upon and smooth." If the electrode be of zinc or of copper, the oxychlorids of those metals are formed, and exercise their chemical effects upon organic matter with which they are brought in contact; hence they act as germicides and caustics. At the negative pole the caustic alkalies form and destroy tissue, if the application be sufficiently prolonged. The effects may be graded according to the current-intensity and duration of the application. Effective electrolysis is excited with 45 milliampères of current.[2]

If the negative pole be applied to the gum over a tooth, and the positive pole to the back of the neck and its position shifted from time to time, alkalies may be liberated in the tissues and cause effects ranging from effective counter-irritation to the actual breaking-down of indurated tissue, or the removal of hyperplasia.

If the positive pole be placed in a root-canal and the negative pole upon some other point, fluids of the tooth or tissues about the apex of the root (if the electrode be carried into them) are decomposed, and the substances formed act upon the tissues. If a zinc electrode be used, the germicidal and caustic zinc oxychlorid is formed, as above noted.

[1] Bartholow's *Medical Electricity.* [2] *Ibid.*

Another important property of the constant current is utilized in dental therapeutics, viz., the power of such a current to cause the passage through an intervening resistance, of substances in solution from the positive toward the negative pole—cataphoresis, or electrical osmosis. This property is well illustrated by an experiment of Morton's. A glass vessel containing a porous septum dividing it into two chambers, has placed in one of the chambers a solution of starch, in the other a solution of iodin. If now the positive pole of a galvanic combination be placed in the iodin solution, and the negative in the starch solution, the iodin is caused to pass more quickly through the septum, and the blue coloration of the iodin reaction with starch at once appears.

Through the aid of such a current, with appropriate electrodes and under proper insulation, solutions of drugs may be caused to pass along the conducting paths of the dentin—*i. e.*, through their tubuli; medicaments may be carried from the surface of the gum into the pericementum, etc. This principle is utilized in the treatment of hypersensitive dentin, for which special apparatus is necessary, so that the voltage may be raised by small fractions, the dental pulp being peculiarly intolerant of electric currents abruptly applied.

Bleaching-agents, hydrogen dioxid solutions, may be quickly driven into the deeper portions of discolored dentin by this means.

With suitable electrodes, cocain solutions may be driven in about the roots of teeth to render the operation of tooth-extraction painless.

Hypodermatic Medication.

Local anæsthetics—cocain, eucain, and tropacocain—are used by the hypodermatic method to render painless the operation of tooth-extraction. Certain precautions should be observed in their use. The field of operation should be sterilized to prevent the entrance of pathogenic organisms to deep parts. The syringe and all of its parts must be carefully sterilized, as must also the solution employed. Cocain solutions require the addition of an antiseptic. Eucain solutions may be sterilized by boiling. The minimum physiological dose of the drug should be employed, suspended in a large volume of fluid. The injection should be made in, not under, the maxillary periosteum. Injections should never be made in the loose tissue of the corium, as annoying swellings result and anæsthesia fails.

One per cent. solution of Cocaine in listerine.

L. Grunbaum

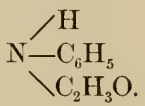
"Items" July 1900

DENTAL PHARMACOPŒIA.

The agents used in dental therapeutics and their modes of action have been considered under the head of dental pharmacology, so that further classification is unnecessary. The therapeutic application of any agent may be determined by noting its description under the alphabetical headings of this section, and then turning to the portion on pharmacology where general and specific group-properties are discussed.

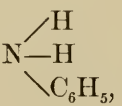
Acetanilid (Antifebrin).

Its name, acetanilid or phenyl acetamide, is derived from its chemical composition, graphically represented in



Acetanilid.

It is an amide, a substitution-product of ammonia, one hydrogen atom of ammonia being replaced by phenol, and one by acetyl, or it may be regarded as anilin,



Anilin.

in which a hydrogen atom is displaced by acetyl. It is a whitish crystalline powder, slightly pungent, without odor; sparingly soluble in water, and freely soluble in alcohol, ether, and chloroform. Combined with ammonium carbonate, the mixture is known as ammonol. Combined with caffein citrate and sodium bicarbonate, the preparation is called antikamnia.

It relieves pain and reduces temperature, and in large doses depresses the action of the heart. The dose is from 3 to 15 grs. In combination with ammonium carbonate its depressing action on the heart is almost neutralized. Used in painful affections, in neuralgia, and to lessen the pains of pulpitis and pericementitis; grs. v., repeated until 15 grs. are taken. It has been used locally as a mild antiseptic to raw surfaces, instead of the usual antiseptic powders.

Acids.

Acid, Acetic, $HC_2H_3O_2$.

The glacial anhydrous acid, $C_2H_4O_2$, is used as a caustic.

Acid, Trichloracetic.—The trichloracetic acid is the form in which

acetic acid is used in dentistry, the three hydrogen atoms of the radical being replaced by chlorin, $HC_2Cl_3O_2$. This is a colorless and very deliquescent substance. It coagulates albumin promptly, hence it is caustic. It is used deliquesced as a caustic to destroy gum overhanging developing lower third molars, and to destroy vital remnants of pulps in the roots of teeth. Diluted, it is used as an astringent in pyorrhœa pockets and to soften the deposits of calculi.

ACID, ARSENIOUS.

The anhydrous arsenious acid, or arsenic trioxid, As_2O_3, a white crystalline powder, is insoluble in cold water, but partially soluble in an excess of boiling water, when arsenious acid, H_3AsO_3, is formed, a feebly acid substance. It is soluble in hydrochloric acid and freely soluble in alkalies.

Combined with a fresh magma of ferric hydrate, the soluble arsenites are converted into insoluble arsenite of iron. This fact is made use of to prevent the absorption of arsenic which has been taken into the stomach. Evacuation of the contents of the stomach should follow.

Applied to tissues, arsenic causes violent inflammation, followed by profound degenerative changes; the application is attended by much pain. The inflammation excited by its presence prevents the absorption of any but a very minute amount. It is used in dentistry for the sole purpose of devitalizing the pulps of teeth. It is made into paste and applied to the pulp; or cotton-fibre is rolled in the paste, dried, and small pieces of it applied.

℞. Acid. arsenosi,
Cocain. hydrochlorid., *or* } āā gr. x;
Morphinæ acetat.,
Ol. cinnamomi, *or*
Ol. caryophylii, q. s. ft. paste.

ACID, BENZOIC, $C_6H_5CO_2H$.

Benzoic acid is prepared by heating gum benzoin; the acid sublimes in pearly white plates. Faintly soluble in water; freely soluble in ether, chloroform, and strong alcohol. Locally applied it is slightly stimulant. It is markedly antiseptic and non-poisonous, hence is a valuable ingredient in mouth-washes.

ACID, BORIC, H_3BO_3,

is slightly soluble in cold water; soluble in strong alcohol and in glycerin. A non-toxic antiseptic; it is useful as an ingredient in

mouth-washes. Combined with sodium sulfite, to evolve sulfur dioxid, SO_2, for bleaching purposes (see Sodium Sulfite).

ACID, CARBOLIC.

Phenylic alcohol, phenyl hydroxid, C_6H_5HO, when pure, is colorless and crystalline; odor is distinctive. Becomes fluid at 95° F. The addition of glycerin renders it fluid. It is soluble in a great excess of water, a 3 per cent. solution being-permanent. It is readily soluble in ether, chloroform, alcohol, glycerin, and the essential oils. It coagulates albuminous matter, hence is caustic; it acts as a germicide in virtue of the same property; locally applied, it is an anæsthetic. Used in dentistry in full strength to obtund the hypersensitivity of dentin, to relieve the pain of pulpitis; as an antiseptic in carious cavities, in putrescent pulps, in root-canals, and in septic pericementitis. Used as a caustic for canker sores, stomatitis ulcerosa.

In 3 per cent. solution it is used as an irrigating antiseptic and to keep sterilized instruments in, prior to using them.

ACID, CHROMIC, CrO_3.

Chromic anhydrid, when deliquesced or dissolved in water, becomes H_2CrO_4, or chromic acid. A powerful caustic, rarely used in dentistry. Has been used as a dentinal obtundent, and as a caustic in sluggish ulcers. Its present uses in dentistry are in $\frac{1}{6}$ of 1 per cent. solution as a hardening fluid for histological work, and in galvanic batteries.

ACID, GALLIC,

does not coagulate (see Tannic Acid), but causes contraction of bloodvessels, hence is not used as a styptic locally, but when the internal administration of a hæmostatic is indicated. Given in pill-form; dose, grs. 2–20.

ACID, HYDROCHLORIC, HCl.

Rarely used in dentistry. Used to supply deficiency of HCl in stomach and to check fermentative processes there. It rapidly decalcifies the hard tissues of the teeth.

ACID, LACTIC, $C_3H_6O_3$.

A syrupy liquid freely soluble in water. It is the acid of fermentative origin which decalcifies the teeth in the progress of caries. It has the power of dissolving fibrinous exudates, and is used, therefore, to remove the false membrane in diphtheria. In 20–50 per cent. solution it is used to soften deposits of calculi in cases of pyorrhœa alveolaris, and as a stimulant astringent to the engorged soft tissues about the parts.

ACID, NITRIC, HNO_3.

The strong acid is used as a caustic application to canker sores. Its application is productive of pain, so that carbolic acid is preferred for this purpose. Used to touch abraded spots of hypersensitive dentin upon the occlusal faces of the teeth. As decalcification results, such spots are to be excavated and filled.

ACID, OXALIC, $C_2H_2O_4$,

should be plainly marked, as its crystals are readily mistaken for those of magnesium sulfate (Epsom salt). Used to liberate chlorin from calcium hypochlorite, in the process of bleaching discolored dentin (Truman).

ACID, PHOSPHORIC.

The ortho-acid, H_3PO_4, in solution, is the fluid ingredient of zinc-phosphate cements.

ACID, SALICYLIC, $HC_7H_5O_3$,

is contained in oil of gautheria (wintergreen). It may be obtained from this source, or be made synthetically by decomposing sodium salicylate with hydrochloric acid. Is slightly soluble in cold, freely soluble in hot water. Soluble in alcohol. Borax in solution aids the solubility of salicylic acid, hence their conjoined use in mouth-washes. In solution strength of 1 : 200, salicylic acid destroys most of the forms of bacteria found in the mouth. Its principal use in dentistry is as an ingredient of mouth-washes.

ACID, SULFURIC, H_2SO_4.

Sulfuric acid is used in dental therapeutics in 50 per cent. solution, mainly to gain access to, to enlarge, and sterilize root-canals so minute as to refuse entrance to fine instruments. It is used full strength as a destructive obtundent in cases of hypersensitive dentin. In weak solution, 10 per cent., it is used to neutralize the free alkali in teeth left after applications of sodium dioxid or sodium-potassium. The same solution is used to soften the deposits in cases of pyorrhœa alveolaris, to remove dead bone, and as an astringent. It is a solvent of the calcium salts of the teeth and destroys organic matter by a process of chemical dehydration.

Acid. sulfuric. aromatic. is a mixture of sulfuric acid in alcohol, spirit of cinnamon, and tincture of ginger, in strength of $7\frac{1}{2}$ per cent. By the action of the sulfuric acid on alcohol a portion of the latter is oxidized, forming an ether. Its uses are those of dilute sulfuric acid, and, in addition, it has marked stimulating properties. It is used full strength in treating pyorrhœa pockets, as a calcic solvent, a germicide,

a stimulant, and an astringent. It is used to dissolve carious bone and to stimulate the vital parts to reparative action.

ACID, SULFUROUS, H_2SO_3,

acts as a bleaching-agent by a process of reduction, seizing upon the oxygen of the pigment. Is used as a bleaching-agent for discolored dentin. It is generated by mixing the dry powders of sodium sulfite and boracic acid, placed in contact with discolored dentin, water is applied and sulfurous acid is disengaged (Kirk). It acts as a deodorant and antiseptic in the same manner as above given.

ACID, TANNIC, $C_{27}H_{22}O_{17}$,

is readily soluble in water, alcohol, and glycerin. It is the astringent principle of most of the vegetable astringents. When oxidized, as when tannic acid is taken into the body, gallic acid is formed. It brings about a rapid coagulation of the blood with a contraction of vessel-walls; hence is used as a styptic locally in powder. In solutions of various strengths adapted to the conditions, it is applied to reduce the passive congestion of swollen gums and tumid mucous membranes. Its solution in glycerin is known as glycerite of tannin; it is made by dissolving 1 part of tannic acid in 4 parts of glycerin, through the aid of heat. This preparation is used in cases of swollen and passively congested mucous membranes; the hygroscopic glycerin attracts the fluid exudates, and the tannic acid causes contraction of the engorged tissues. Tannic acid, in combination with alum, glycerin, and thymol, is used to mummify, and harden the remnants of pulps not removable by mechanical means.

ACID, TRICHLORACETIC. (See Acid, Acetic.)

Aconite.

Tincture of the root, tinc. aconiti radicis; dose 1 to 5 drops; used in one-drop doses to reduce the pulsations of an overacting heart in acute inflammations. Locally used in combination with iodin to subdue pericemental inflammation:

℞. Tr. iodin., } āā.—M.
Tr. aconiti rad., }
Sig. Painted on gum, over the affected tooth.

In old preparations the evaporation of the alcohol may, by concentration of the solution, increase the volume-strength, and poisoning may occur if used freely. Antidotes are cardiac stimulants—ammonia, whiskey, brandy. The physiological antidote is atropia.

Aconitine

is the alkaloid of aconite, an extremely active poison. Its dose is $\frac{1}{100}$ gr. Its dental use is in ointment:

℞. Aconitini, gr. j;
Cerat. simp., ℥j.—M.
Sig. To be rubbed over the tissues in front of the ear to relieve the trismus-like pains which may attend difficult eruption of lower third molars.

Alcohol.

Ethylic alcohol, C_2H_5HO, or ethyl hydroxid, 95 per cent. alcohol: being about 94 per cent. by volume, 91 per cent. by weight of ethyl hydroxid—*i. e.*, 9 per cent. by weight, 6 per cent. by volume of water. Specific gravity at 60° F. 0.820.

Methyl alcohol, CH_3HO, methyl hydroxid. Whiskey and brandy containing from 48 to 56 per cent. by volume of ethyl alcohol, are used as diffusible stimulants in cases of syncope from any cause; dose, ℥ss–j. Strong alcohol is very astringent, and is antiseptic. Being hygroscopic, it readily takes up water; this property is utilized in producing dryness of the dentin prior to inserting fillings and canal-fillings. Both ethyl and methyl alcohols are used as solvents for several vegetable gums which are employed in both laboratory and office uses. (See Lining Varnishes.)

PHENYL ALCOHOL, C_6H_5HO, PHENYL HYDROXID. (See Carbolic Acid.)

Alum.

Aluminum potassium sulfate, $Al_2(SO_4)_3,K_2SO_4 + 24H_2O$.
Aluminum ammonium sulfate, $Al_2(SO_4)_3,(NH_4)_2SO_4 + 24H_2O$.

It is the potash-alum—aluminii et potassii sulfas—which is usually employed in medicine. It is a colorless salt, soluble in about fifteen volumes of cold water, and in three-quarters of a part of hot water. It has a sweetish, astringent, and acid taste, but unless the specimen contain free acid, alum solutions should be neutral in reaction. Alum-exsiccatum—dried alum—is alum whose water of crystallization has been driven off by heat, forming a white granular powder.

Solutions of alum are used as astringent washes in cases of passive hyperæmia of the gum-tissue, such as those caused by the presence of salivary calculi, and in pyorrhœa alveolaris. It should be ascertained that the alum contains no excess of acid—that is, should not redden blue litmus paper—before it is used. Powdered alum is a powerful

styptic, and may be used on cotton tampons for the relief of alveolar hemorrhage. Alum exsicatum is used as a mild caustic to exuberant granulations: it acts both as a caustic and astringent.

Ammonium.

Ammonium hydrate, NH_4HO, and the **neutral carbonate** $(NH_3)_2CO_3$, are used in combination with oils of lemon, pimento, and lavender, in solution of alcohol and water as a diffusible stimulant, under the name of spiritus ammoniæ aromaticus. Given in doses of ʒss–ij, it acts as a prompt cardiac stimulant.

Ammonium nitrate, NH_4NO_3, is the salt from which the anæsthetic, nitrogen monoxid (nitrous oxid), is disengaged.

$$NH_4NO_3 + \text{heat (350°–450° F.)} = N_2O + 2H_2O.$$

Ammonol (see also Acetanilid)

is a combination of acetanilid with ammonium carbonate; the latter is added to neutralize the depressing effect of acetanilid upon the heart. Dose, grs. v–x. Used in neuralgic conditions, hemicrania, tic douloureux, and to benumb the pains of pulpitis and acute pericementitis. It is frequently very effective in the latter affection.

Amyl Nitrite, Amyl Nitris, $C_5H_{11}NO_2$

Dose, ♏ij–v. It is dispensed in glass pearls containing these amounts. It is administered by inhalation. It depresses the inhibitory apparatus of the heart; used when the peripheral bloodvessels are in a state of marked contraction, it causes their immediate dilatation; hence is used in angina pectoris, to relieve the spasmodic contraction of the heart and vessels, and in conditions of shock with pale surface. It has been suggested as an antidote to chloroform, when the conditions named present, but as it is also the antagonist of strychnia, the antidote of chloroform, its use must be guarded.

Antipyrin

is phenyl-dimethyl-pyrazolon. Irrespective of physiological experiments, a substance having the chemical composition given would, at the present day, be known to have the power of reducing pain and lessening temperature, which are in fact its uses. Dose, iii.–x grs. Antipyrin is freely soluble in water. Small doses decrease, large doses increase, the reflex activity of the brain,[1] the cause of the depression being due to sedation of the sensory nerves and of their centres in the spinal cord. Applied to mucous membranes or beneath the skin, this agent is a power-

[1] Hare, *Practical Therapeutics.*

ful local anæsthetic, the anæsthesia lasting often for several days. Bartholow[1] and Hare both agree that the cardiac depressing action of antipyrin is over-rated, being only marked in the cumulative action of the drug. Its antidotes are atropia and stimulants. It is used to relieve the pain of facial neuralgia, pericementitis, and acute pulp-diseases.

Aristol, Dithymol—Di-iodid.

Introduced as a substitute for iodoform, upon the assumption that iodoform acts as an antiseptic in virtue of setting free iodin when brought in contact with vital tissues; aristol containing twice the amount of iodin, should have greater power; the thymol should also exercise particular antiseptic action. Reports as to its efficacy are contradictory. Upon cocci and bacilli aristol has less power than iodoform (Hare). It is used in dentistry as a dressing in pulp-canals which have contained putrescent pulps; its value as a persistent antiseptic in these cases is masked, owing to the previous or simultaneous use of other antiseptics.

Borax, $Na_2B_4O_7 + 10H_2O$. Boric Acid.

Impure borax is the common flux of the dental laboratory. In this state it sometimes contains borates of other metals than sodium, which may cause contamination of the noble metals (Hiorns). Pure borax is soluble in twelve parts of water. Saturated solutions are used as mouth-washes in conditions of stomatitis (aphthæ) as an antiseptic. It is an ingredient of Dobell's solution, which is used as an antiseptic wash in catarrh of, and empyæma of the antrum, and in stomatitis:

℞. Sodii boratis, Sodii bicarbonatis, āā ʒj;
Acidi carbolici, gr. xxx;
Glycerni, ʒj;
Aquæ puræ, Oij.—M.
S. Used warm, as a spray.

Boric acid is a useful addition to antiseptic mouth-washes. Used dry, on aphthous sores, and in cases of cancrum oris, ulcerative stomatitis, etc., it is an admirable antiseptic. An ointment:

℞. Ac. boric., ʒj;
Cerat. alb., ʒj;
Paraffin, ʒij;
Ol. amyg. exp., fʒij.—M.

[1] *Materia Medica and Therapeutics.*

is a useful application to chapped lips and in herpes labialis, and to prevent chapping of the hands.

Boroglycerin [1]

is made by mixing 62 parts of boric acid with 92 parts of glycerin, in a tarred porcelain capsule, at a temperature of about 300° F. The acid is added to the glycerin gradually, stirring constantly ; when the mixture is reduced to 100 parts it is poured on a block slightly wet with petroleum ; when dry it is cut in blocks and kept in stoppered bottles. One ounce of these blocks is added to one ounce of glycerin to form glycerite of boroglycerin, a vehicle for carbolic acid and other substances applied as mouth-washes.

Bromids.

Potassium bromid, gr. v–3j.

Sodium bromid, gr. v–3j.

Used as sedatives when the cerebral circulation is overfull, as in teething children and in acute pericementitis. Applied locally to reduce the excessive irritability of the mucous membrane of the soft palate and fauces. Will relieve the form of migraine accompanied by flushed face and injected eye. Will quiet the irritability of hysterical dental patients.

Caffein.

Caffein, the active principle of coffee ; thein, the active principle of tea, and that of guarana, are chemically identical. Used in combination with acetanilid in the mixture called antikamnia (which see) to counteract its depressing and increase its analgesic effects.

Cajuput Oil. (See Oils, Antiseptic.)

Calcium.

Calcium oxid (lime) in solution in water has been used as an antacid mouth-wash.

Calcium carbonate (precipitated chalk) is used as an ingredient of dentifrices, and as an antacid, rubbed over the teeth at night, to neutralize the acids causing caries and erosion, and to lessen the hypersensitivity of dentin.

Calcium hypochlorite in mixture with calcium chlorid, is used as a bleaching-agent for discolored dentin. Chlorin is liberated by the action of dilute organic acids.[2]

The hypophosphites, phosphates, and lacto-phosphates have been administered to increase the amount of calcium salts in dentin in which

[1] Hare, *Practical Therapeutics.*

[2] Truman.

these salts were supposed to be deficient; it has never, however, been demonstrated that they have any effect in this direction.

Calcium sulfate (gypsum) calcined, is plaster of Paris.

Camphor.

Camphor liniment (soap, camphor, oil of rosemary, alcohol, and water) is used with friction over the masseter muscle to relieve the temporary spasms caused by difficult eruption of lower third molars.

Camphor spirits, a few drops in a glass of water, is used to lessen the irritability of the soft palate and fauces, to permit manipulation of those structures.

Campho-phenique.

A fluid substance formed by the combination of nearly equal parts of carbolic acid and gum camphor, said to be a definite chemical body having the formula $C_8H_{11}O$, the reaction presumably being

$$\underset{\text{Camphor.}}{C_{10}H_{16}O} + \underset{\text{Phenyl alcohol.}}{C_6H_5HO} = 2C_8H_{11}O.$$

It possesses the antiseptic and anæsthetic properties of carbolic acid, and, in addition the stimulant property of camphor. It does not produce an eschar; it is insoluble in water. Its uses are those of carbolic acid in general; but it has not the depressing effect of that agent upon the vitality of tissues to which it is applied. It is used in full strength for hypersensitivity of dentin, to sterilize infected and purulent pulps, and as a stimulant antiseptic application to the walls of chronic alveolar abscesses. It may be used as a stimulating antiseptic application to pyorrhœa pockets after removal of the deposits and washing with hydrogen dioxid.

Cantharides (Spanish Flies).

An active principle called cantharidin, is extracted from the crushed bodies of the beetle, cantharis vesicatoria. It is used in plaster, charta cantharadis, or in collodion, collodion cum cantharide, to produce blistering. A blister placed in front of the ear or beneath the ear is a counter-irritant of service in relieving the pains of pulpitis and pericementitis, after local therapeusis has been applied. A small blister applied to the gum, at some distance from an affected root, is useful in cases of sluggish pericementitis of the chronic type.

Capsicum.

Depending upon the strength in which they are used, preparations of capsicum are stimulants or irritants, and are used to stimulate sluggish local circulation or as counter-irritants.

Tr. capsicum may be used alone, or as tr. capsici et myrrhæ; either is added to water until cloudiness appears; used as a stimulant wash in cases of atonic affections of the gum-tissues, and to hasten the separation of sequestra of bone. Powdered capsicum and ginger, made into packet-form, in small muslin bags, and called capsicum bags, are useful as counter-irritants in acute pulp-affections and in chronic pericemental disturbances.

Chloral Hydrate, $C_2HCl_3O.H_2O$.

The syrup of chloral, in doses of f3j, is an admirable hypnotic in cases of insomnia from pulpitis, and after arsenical applications have been made to a pulp. In saturated solution it has been used to reduce the hypersensitivity of dentin. Its solutions are markedly antiseptic and are irritant to soft tissues.

Chloroform, Formyl Trichlorid, $CHCl_3$.

The most dangerous of the general anæsthetics in dental practice; it should never be used in dentistry for this purpose. The list of fatalities recorded from the employment of chloroform in tooth-extraction is a most formidable one. The semi-erect position adds to the danger always accompanying the use of this agent. It is used in dentistry as a counter-irritant. A piece of blotting-paper saturated with chloroform laid upon mucous membrane or skin, and its evaporation prevented by covering with rubber cloth, acts as a stimulant, counter-irritant, or vesicant, according to the length of application. It is used as a solvent for gutta-percha base-plate, to make the root-filling solution called chloropercha.

Cobalt.

A powder named cobalt has been used to destroy the vitality of the dental pulp, particularly after the Herbst method. Analysis has shown this powder to be metallic arsenic or arsenic sulfid (Kirk).

Cocain (Hydrochlorid), Benzoyl-methyl Ecgonin.

Decomposed, by boiling, into methylic alcohol, benzoic acid, and ecgonin; therefore, unlike eucain, its solutions cannot be sterilized by boiling. Solutions of cocain paralyze the terminals of both sensory and special sense-nerves with which they are brought in contact. Applied to nerve-trunks they prevent the transmission of sensory impressions. Death from overdose is due to paralysis of respiration. Poisoning should be treated with stimulants—ammonia, strychnia, or ether.

It is used in dentistry in solutions of various strengths as a local anæsthetic. In from 4 to 10 per cent. solution as an application to the

mucous membrane to render incision painless. In saturated solutions or in crystals, or in paste with glycerin, is used in acute diseases of the pulp as an analgesic. In solution with agents added to overcome ill-toward effects, and to prevent the growth of organisms which will develop in watery solutions of the drug, cocain is used as an agent for hypodermatic injection to render the operation of tooth-extraction painless.

℞. Cocain. hydrochlorid.,	gr. $\frac{1}{8}$;
Morphinæ sulph.,	gr. $\frac{1}{12}$;
Atropinæ sulph.,	gr. $\frac{1}{150}$;
Trinitrin (1 per cent. solution),	gtt. j;
Acid. carbolic.,	gtt. j;
Aquæ,	q. s. ft. ʒss.—M.

The above is a full dose, making about one-half syringeful. The syringe-needle is passed deep into the connective tissue between mucous membrane and alveolar periosteum over a root, and several drops of the solution are injected. A similar injection is made upon the opposite side of the same root, or over other roots; extraction should be done almost immediately. Prior to making the injection the mucous membrane at the point of injection should be carefully sterilized, as should also the syringe and needle.

In strong solution, cocain is forced into the dentinal tubuli and into the dental pulp by means of the cataphoric current, to obtund hypersensitive dentin or to paralyze the pulp and permit its removal. Cocain without this driving force is ineffective as a dentinal analgesic, even in saturated solution or in glycerin paste.

Collodion.

A solution of pyroxylin (gun-cotton) in ether and alcohol. Exposed to the air, the solvent evaporates and leaves a thin, transparent, impermeable film. Cantharidal collodion is formed by adding cantharides-extract to collodion. Styptic collodion is made by dissolving tannic acid in collodion: tannic acid, grs. 20; collodion, ʒj. This makes a useful application to oozing, abraded surfaces after previously sterilizing them. Steresol (which see) varnish is, however, superior to it.

Copper, Cuprum.

Black cupric oxid combined with orthophosphoric acid forms a black cement, orthophosphate of copper, advised as a filling-cement in dentistry to underlie other materials.[1] Cupric sulfate—bluestone—according to

[1] Ames.

the strength of solution in which it is used, is an astringent or caustic. Copper sulfate acts as a germicide, like the other metallic salts mentioned, by causing coagulation of albumin.

Miller's[1] experiments demonstrated that crystals of cupric sulfate placed upon the surface of a pulp transformed the entire pulp into a green, antiseptic, tough mass. It causes, however, a green discoloration of the dentin, and if used when putrefactive decomposition is in progress, black copper sulfid is formed by the action of the hydrogen sulfid present.

A crystal of cupric sulfate is a useful application to the dentin of deeply infected teeth which are to be enclosed by barrel crowns. In strong solution it is a useful agent in the cauterant treatment of pyorrhœa pockets. In 1 per cent. solution it is a useful astringent and antiseptic wash for relaxed conditions of the soft tissues about the mouth.

Cotton.

The hairs of the seeds of the cotton-plant (gossypium herbaceum). In the carded state it is the raw cotton of dentistry. Boiled with a 5 per cent. solution of sodium or potassium hydrate, fatty substances and foreign matters are washed away, and the boiled cotton is afterward washed with calcium hypochlorite; this is absorbent cotton. Each fibre consists of elongated tubular cells, which absorb any moisture with which the cotton is brought in contact. The cotton should contain no free alkali or free acid. Mixed with arsenical paste and dried, it constitutes devitalizing fibre.

Corrosive Sublimate. (See Mercuric Chlorid.)

Creosote.

A product of the distillation of wood, etc.; it contains carbolic acid and allied substances; its properties and uses are those of carbolic acid.

Cresols, nominally $C_6H_4CH_3OH$.

methyl phenol.

Obtained by fractional distillation of crude carbolic acid at 365°–401° F. There are three cresols, ortho-, meta-, and para-cresol, the second being the most powerful germicide. Para-cresol is a local analgesic, destroying the sense of pain, but not tactile sensibility.[2] It will be seen that the chemical composition of these substances shows them to be allied to cocain and carbolic acid. Frankel and Gruber found a mixture of the three cresols—called trikresol—to possess three times the disinfectant power of carbolic acid. Trikresol may be used with advantage to replace carbolic acid in dental practice.

[1] *Dental Cosmos.*

[2] McNeill, *Edinburgh Med. Jour.*, 1886.

Ergot.

The fluid extract in drachm doses is used as a hæmostatic.

Erigeron, Fleabane.

The oil of erigeron, in doses of from twenty to thirty drops in capsules, is used in oozing alveolar hemorrhage after the extraction of teeth.

Ether, Ethyl Oxid, $(C_2H_5)_2O$.

Used as the major anæsthetic for dental operations, when it is essential to keep the patient anæsthetized for comparatively long periods, as in the removal of imbedded, impacted teeth. Dividing the anæsthetic stages of ether into four, the first stage is when sensibility to pain is dulled before the loss of consciousness occurs; teeth may be extracted in this period, or excessively hypersensitive dentin may be cut, without pain.

Ethyl Chlorid, C_2H_5Cl,

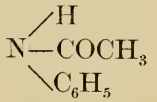
is too volatile for use as a general anæsthetic. It boils at 12° C. It is used as a refrigerant local anæsthetic, its rapid evaporation causing quick paralysis of sensory nerve-terminals or trunks. A spray of this agent directed against vital dentin will benumb its sensitivity, and if the application be prolonged refrigeration extends to the pulp, which in many cases can be extracted painlessly. A spray directed upon the gum will freeze it and permit the extraction of a tooth without pain.

Eucain Hydrochlorid,

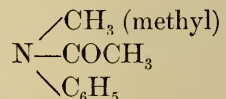
a synthetic substance, having a methyl-benzoyl basis, as in cocain; its composition is, however, much more complex than that of the latter. Solutions in water may be boiled without decomposition; hence, unlike cocain, solutions may be sterilized by boiling. It is not so toxic as cocain. It may be used in watery solutions which have been boiled, in amounts of $\frac{1}{2}$–$\frac{3}{4}$ gr. by hypodermatic injection, to render tooth-extraction painless.

Exalgen, Methyl-acetanilid,

is acetanilid in which the remaining hydrogen atom of the basal ammonia is replaced by methyl:



Acetanilid



Methyl acetanilid, or exalgin.

Its analgesic power is greater than than of acetanilid. Its uses are the same in treating painful affections, but it is not used as an antipyretic.

Formalin,

a 40 per cent. solution of the gas formaldelyd (CH_2O) in water. Formaldehyd is an oxidation-product of methyl alcohol. A powerful antiseptic; in 1 : 1000 solution it prevents the growth of anthrax spores; in 1 : 2000 solution it checks putrefaction in bouillon.[1] The vapor (powerfully antiseptic) is given off from formalin solutions at ordinary temperatures. It produces tough coagula when brought in contact with albuminous substances. Both vapors and solutions are very irritating. Any strength in excess of 5 per cent. placed in root-canals is productive of irritation; and the dental pulp responds painfully to any solution of a strength above 3 per cent.[2] It is used in the indicated strength in connection with putrescent pulps, prior to attempts at mechanical manipulation, to penetrate and sterilize to the root-apex. In 1 per cent. solution it is an excellent injection for deep-seated abscesses, particularly those of a chronic type. In 1 per cent. solution in combination with other agents it is an admirable antiseptic mouth-wash.

Glycerin, Propenyl Alcohol, $C_3H_5(OH)_3$,

is set free when natural fats are boiled in a caustic alkali. Stearates of potassium or sodium (soaps) are formed and glycerin is liberated. It has moderate antiseptic powers and is very hygroscopic. A drachm or two injected into the rectum produce a watery stool, and this fact is utilized in the derivatant treatment of teething-convulsions. Mixed with equal parts of water it is useful to relieve "dry mouth and fauces." In combination with tannic acid it forms glycerite of tannin (which see).

Guaiacol.

A distillation-product of creasote, containing from 60 to 90 per cent. of that substance. Its uses are in general those of creasote. A 10 per cent. solution of anhydrous cocain hydrochlorid in pure guaiacol is termed guaia-cocain,[3] and is used instead of watery solutions of cocain in connection with the cataphoric current to benumb hypersensitive dentin.

Hamamelis.

Fluid extract, dose internally 5–20 drops. The distilled extract (Pond's extract) may be used in doses of ʒss–ʒj internally. Internally it is used as a hæmostatic to check oozing hemorrhage. Locally it is used to reduce vascular engorgement. It is an efficient antiphlogistic for use in inflammatory conditions of the mouth and fauces. Is very useful as a general mouth-wash in cases of pericementitis, after the excit-

[1] *U. S. Marine Hospital Reports*, July, 1897. [2] L. Jack.

[3] W. T. Morton.

ing causes of the inflammation have been removed. It is used as a wash in cases of gingivitis.

Hydrogen Peroxid, Hydrogen Dioxid, H_2O_2.

In 3 per cent. watery solution yielding ten volumes of oxygen. In 5 per cent. ethereal solution. In 25 per cent. ethereal solution, caustic pyrozone. Sodium dioxid, from which hydrogen dioxid is disengaged by the action of dilute mineral acids. Most of the watery solutions of hydrogen dioxid are slightly acid in reaction. Solutions of hydrogen dioxid deteriorate with age, and very quickly upon open exposure. Their activity may be judged by the violence of the reaction which occurs when potassium permanganate is added to the solutions. Used as a germicide, antiseptic, and disinfectant; acting by virtue of the nascent oxygen set free when the solutions are brought in contact with organic matter. (See Antiseptics, Nascent Oxygen, and Caustic Alkalies.) Used in abscess-cavities, pus-pockets, pulpless roots, pulp-diseases, and in all conditions where an active and non-toxic antiseptic is indicated.

Iodin, Iodids.

Tr. iodin and liq. iodi comp. (Lugol's solution). Tr. iodin evaporated to one-fourth its volume is called dental tincture of iodin.[1] Iodin is incompatible with mineral acids, metallic salts, and vegetable alkaloids. Iodin in vapor, solid, or in solutions, decomposes hydrogen sulphid and hydrogen phosphid, acting as a deodorant. It combines with albuminous substances, acting as a germicide. Locally applied, it acts as a stimulant, irritant, or caustic, depending upon the concentration of the solution. It is used as a counter-irritant in cases of pulpitis and pericementitis (acute), and to resolve indurations about the teeth due to chronic inflammation of the pericementum. Used in pulp-chambers to quickly deodorize the putrescent pulp. If the cavity be washed with ammonia-water, afterward any iodin-stain is removed and the dentin whitened. Used diluted in conditions of tumid gums, and as a stimulant application in deep pyorrhœa pockets. The liq. iodi comp. is preferable in such cases.

Iodoform, Formyl Tri-iodid, CHI_3.

Iodoform, by virtue of its organic radical, is a local anæsthetic. It is antiseptic, although not a germicide. Its action in this direction is believed to be due to one of two causes: either by virtue of the iodin which is set free when iodoform is brought into contact with infected tissues, or to chemical changes which it induces in the poisonous prod-

[1] Flagg.

ucts of bacteria, rendering them non-toxic. The reduction of irritability by the formyl radical must, in addition, play an important part in inducing regeneration of tissues over surfaces to which iodoform is applied. Used in conjunction with arsenic trioxid in devitalizing paste, it lessens the pain incidental to pulp-devitalization.[1]

℞. Acid. arsenosi,	gr. v ;
Iodoformi,	gr. x ;
Ol. cinnamomi,	q. s. ft. paste.

There is much discrepancy of opinion as to the value of iodoform, brought about by the observation that it is not a germicide ; nevertheless in pyogenic conditions of a chronic type it appears to have distinct value. It is used in solution or in powder in the cavities of chronic abscesses or pyorrhœa pockets. Gauze charged with iodoform, iodoform-gauze, is used to pack abscess-cavities which have been opened artificially through the alveolar walls, and after root-amputations. Under its use regeneration and healing proceed more uniformly than with most medicinal applications. Iodoform is used freely in cases of tuberculosis of the jaws.

Iodol, Tetra-iod-pyrrhol, C_4I_4NH.

Introduced as a substitute for iodoform, it contains about the same volume of iodin in looser combination. It is inodorous. Applied to wounds iodin is set free and acts upon albuminous substances, from which ozone is disengaged, which oxidizes compounds of sulfur and phosphorus.[2] Its uses are those of iodoform, except in arsenical pastes, where the anæsthetic property of iodoform is utilized.

Iron.

Freshly prepared hydrated sesquioxid of iron is the antidote to arsenic. Tr. ferri chlor. is occasionally used as a styptic in alveolar hemorrhage. Monsell's solution, sol. ferric subsulfate, is a powerful styptic, but may cause sloughing of tissues. Preparations of iron are rarely used in the mouth on account of the stains they produce upon the teeth.

Kino, Krameria,

vegetable astringents used in mouth-washes, acting by virtue of the tannic acid contained in them.

Lysol

is a carbolized compound, made by dissolving in fat and saponifying with alcohol that part of coal-tar which boils between 190° and 200° C.

[1] Truman. [2] Bartholow.

It forms a clear, soapy fluid with water. Is used in 2 to 4 per cent. solution to sterilize the washed hands, and in boiling solutions of the same strength to sterilize instruments. In full strength it is an excellent penetrating antiseptic as a first application to pulps in a state of partial putrescence.

Magnesium.

Magnesium hydrate in suspension is Phillip's milk of magnesia. It is perhaps the best of all antacids for use in dental therapeusis. It forms a film of magnesium hydrate upon the surfaces of the teeth and aids materially in checking the advance of dental erosion.

Magnesium sulfate in doses of ℥ss, well diluted, is an excellent derivative saline cathartic in cases of acute pericementitis.

Menthol, Mint Camphor.

The active principle of peppermit, upon which its effects depend. It is a local anæsthetic and produces contraction of the small vessels of the part to which it is applied. This combination of properties renders it very useful in treatment of acute pulpitis. Used dissolved in chloroform or oil of cassia, it is an excellent agent in the treatment of the pericemental irritation which occasionally follows upon the removal of the pulp. It is pumped in the canals and permitted to remain for a day or longer. A solution (gr. j–℥) is a useful wash in cases of sluggish stomatitis.

Mercury.

Metallic mercury is used as the solvent of dental alloys.

Mercury bichlorid, $HgCl_2$, in strength of 1 : 2000[1] is found to act as an effective sterilizing agent in the human mouth, killing in a very few minutes, nearly all forms of bacteria found in that cavity. Its disagreeable taste and the danger of discoloring the dentin of the teeth contraindicate its use as a general oral antiseptic. In pulpless teeth, particularly in those in which the pulp has become gangrenous and undergone putrefactive decomposition, the use of mercuric chlorid is contraindicated because of its reaction with the hydrogen sulfid generated during this type of fermentation. The danger of discoloring, salts of mercury being formed and finding their way into the dentinal tubuli, is an ever-present one.[2]

It has been found that tablets composed of mercury bichlorid and thymol in equal parts, when crushed in the base of a pulp-chamber against the stumps of pulps which have been intentionally devitalized, will maintain a prolonged antiseptic condition in the roots of teeth so treated.[3]

[1] Miller, *Micro-organisms of the Human Mouth.*

[2] Kirk's *Operative Dentistry*, chapter "Bleaching."

[3] Miller, *Proc. Columbia Dental Congress.*

Corrosive sublimate has found but limited clinical application in dental practice, it being possible to induce antisepsis with other germicides which have not the disadvantageous features of the mercury salt. It is not adapted for sterilizing metallic instruments on account of its corrosive action upon steel, and the precipitation of mercury upon other metals. In hot 1 : 2000 solution it is an effective sterilizer for glassware used by the dentist. In 1 : 1000 solution it is an effective lavage in conditions of ulcerous stomatitis, although hydrogen dioxid followed by potassic chlorate has largely displaced it for this purpose. A 1 : 1000 solution in hydrogen dioxid is an excellent germicidal application to abscess-walls.[1]

Mercury sulfid, HgS, vermilion, is the pink coloring-matter of gutta-percha base-plate. As this salt is insoluble in lactic acid, fillings made of pink gutta-percha do not acquire a rough surface like those made of preparations containing the soluble zinc oxid. The red oxid of mercury is used as an ingredient for lip-salves to relieve cracks and abrasions about the lips :

R.	Mercuric oxid.,	ʒj ;
	Tr. benzoin,	ʒj ;
	Cerat. simp.,	ʒij ;
	Liq. potassii,	gtt. ij.—M. et ft. ungent.

(J. F. Flagg).

Methyl Chlorid, Mono-chlor-methane, CH_3Cl,

the lightest and most volatile of the compounds of chlorin with methane, the heaviest being chloroform, tri-chlor-methane, $CHCl_3$. Methyl chlorid vaporizes at a temperature below zero ; hence it is the most active refrigerant available as a local anæsthetic. It possesses an additional advantage over ethyl chlorid, in that it is but slightly inflammable. A spray of methyl chlorid directed against the gum for a fraction of a minute or longer will create sufficient local anæsthesia to render the operation of tooth-extraction painless. The application should not be continued too long, or tissue-death will result. A spray directed against hypersensitive dentin or an exposed pulp, will render both entirely anæsthetic. Vital pulps may be rapidly paralyzed through the application of a spray of methyl chlorid. The spray may be used as a cold test to determine the vitality of a pulp.[2]

Morphia.

The anodyne alkaloid of opium. It possesses the power of benumbing the functions of the sensory nervous tract when internally admin-

[1] Rhein. [2] *Ibid.*

istered. Locally applied it is an obtundent to sensory nerve-terminals. The sulfate is the most effective salt for internal use; the acetate for external application. Morphinæ sulfate, gr. $\frac{1}{8}$, administered a half-hour before operating will in many cases so benumb sensitivity that the pain of cutting hypersensitive dentin is materially reduced. The same dose may be required as a general anodyne in conditions of pulpitis and acute pericementitis; it has been largely displaced for such purposes by the coal-tar derivatives, which relieve pain without the unpleasant after-effects frequently following upon the use of morphia—*i. e.*, constipation and headache.

Acetate of morphia was the usual anodyne ingredient in arsenical paste-formulæ; it has been almost entirely superseded by cocain hydrochlorid.

Naphthalin, $C_{10}H_8$.

The naphthalin derivatives are all analogues of carbolic acid. Resorcin (which see) belongs also in this chemical and medicinal group. Carbolic acid and resorcin belong in the group which has but a single benzene nucleus, C_6H_6: naphthalin in that having a double benzene nucleus. Phenylic alcohol is benzene in which one hydrogen atom is replaced by hydroxyl, C_6H_5HO. Resorcin has two of its hydrogen atoms so replaced, $C_6H_4(HO)_2$.

Naphtol.

The hydroxyl derivative of naphthalin has the composition $C_{10}H_7HO$. There are two naphtols, the alpha and the beta, both having the same formula—are isomeric. The latter is more soluble in hot water, and its leaflet-like crystals have lower melting- and boiling-points than the needle-like crystals of α-naphtol. Only β-naphtol is used in surgery.

Hydronaphtol, a proprietary agent, is said to be identical with β-naphtol.[1] The penetrating and preserving qualities of this agent, however, appear to be superior to those of β-naphtol.

Naphtol solutions are made in alcohol, these solutions being miscible in hot water. Miller's experiments indicate that these solutions are promptly germicidal,[2] but that they do not induce prolonged antisepsis.[3]

The chemical analogy of naphtol to carbolic acid is, of course, an indication of some or of a close degree of physiological properties.

β-naphtol or hydronaphtol in 1 : 300 solution is used as a spray for sterilizing pyorrhœa pockets, for sterilizing alveoli after extraction and prior to plantation operations. In 1 : 50 solution or stronger it is used in septic pulp-canals, and in the treatment of septic apical pericementitis.

[1] Gould, *Illustrated Dict. of Medicine.* [2] *Micro-organisms of the Human Mouth.*
[3] *Dental Cosmos*, 1891.

Nitrous Oxid, Nitrogen Monoxid, N_2O.

The only entirely safe general anæsthetic. Anæsthesia is induced in from one-half to two minutes, and ceases in from one to three minutes. Ill-effects from its use are rare; those that have been noted were usually in patients whose vessels were atheromatous. Patients having pulmonary emphysema, or fatty heart, may be distressed for hours after its administration, and its use in the three classes of disorders named cannot be regarded as without danger.

Oils, Antiseptic.

Several of the essential volatile oils are used in dental practice as antiseptics. In addition, most of them possess obtundent action upon the dental pulp. The several oils differ as to their activity in both of these directions. All of these oils belong to the aromatic series—*i. e.*, they have a relationship to benzene.

The oil of caryophyllum (cloves) contains an oxygenated oil. Eugenol, having basic properties, is eugenic acid. The oils of cajuput, cassia, and cinnamon (cassia being the Chinese cinnamon, the Ceylon oil that of cinnamon proper), eucalyptus, gaultheria, myrtle, and thyme, all find useful application in dentistry. Of these, oil of wintergreen possesses the least antiseptic properties, oil of eucalyptus the least obtundent action; the most powerful antiseptic action being in the oils of myrtle, cinnamon, and thyme, the last named being the most marked antiseptic and obtundent. It owes its efficiency to a stearopten, thymol (which see), contained in it. These oils may be applied freely to exposed and aching pulps, benumbing them quite promptly and apparently having no deleterious action upon their vitality.

Oil of cinnamon is a persistent, and powerful, slowly diffusing antiseptic. It appears to preserve stumps of pulps with which it is placed in contact. This oil has wide application in the treatment of septic pulp-canals and their chronic sequelæ.

Oxygen.

Oxygen in gaseous form is combined with nitrous oxid (method of Hewitt) to reduce or prevent the occurrence of the lividity due to a deprivation of oxygen, when nitrous oxid is administered alone. Nascent oxygen is disengaged from compounds in which it is loosely combined, as the dioxids of hydrogen and sodium (see Hydrogen Dioxid), to act as a germicide, disinfectant, and bleaching-agent. Liberated from these compounds in contact with decomposing organic matter, the nascent oxygen seizes upon the hydrogen of such substances, effecting their decomposition. Nascent oxygen quickly destroys the vitality of bac-

teria. Uniting with the hydrogen of staining substances, it effects a bleaching by their decomposition.

Paraffin.

Specimens of hard paraffin, those having high melting-points, are used either alone or combined with other substances, as iodoform or aristol, as canal-fillings.

Phenacetin, $C_{10}H_{13}NO_2$ or $C_6H_4(C_2H_5O)NH,C_2H_3O$.

Acetphenetidin or phenylacetin is a tasteless coal-tar derivative having greater antipyretic and analgesic powers than acetanilid. Dose, iij–x grains.

Plumbum, Lead.

The acetate, in the form of liq. plumbi subacetatis, is the basis of lead-water. It is an astringent antiphlogistic, producing contraction of bloodvessels of parts to which it is applied. It is a useful sedative antiphlogistic in cases where the inflammation from alveolar abscess has invaded the tissues of the cheek. It is applied externally. Internally this solution is actively poisonous.

The following is the well-known lead-water and laudanum mixture:

℞. Liq. plumbi subacet., ℥iv;
Tr. opii, ℥j;
Aquæ, Oj.—M.
Sig. Applied on compresses to the inflamed parts.

Potassium.

Several potassium salts, and also the metal itself, are employed in dental therapy. The metal is used in alloy with metallic sodium to effect the quick decomposition of the products of putrefaction in pulp-canals. It seizes upon the hydroxyl elements of these substances, forming potassium and sodium hydroxids, which in their turn act upon the canal-contents, converting them into innocuous and soluble substances. Applications of potassium sodium (kalium-natrium) should precede all attempts at tooth-bleaching, as the bulk of the offending material is thus removed, permitting the passage of nascent oxygen or chlorin to the deeper parts of the discolored dentin.[1]

Potassium bromid in doses of three grains is a useful remedy to lessen the reflex cerebro-spinal irritation of children due to teething. In cases of convulsion it is administered by the rectum in combination with chloral hydrate, suspended in starch mixture. A full dose, grs.

[1] Kirk.

xx–xxx, will in many cases relieve the wakefulness accompanying painful dental affections.

Potassium carbonate in saturated solution in glycerin is a powerful obtundent of hypersensitive dentin. It is also antiseptic, destroying the putrid contents of pulp-chambers.

Potassium chlorate in strong solutions is an effective agent in all forms of stomatitis. It is particularly valuable in mercurial stomatitis.

Potassium hydrate, as a germicide and disinfectant, has been described above.

A mixture of potassium hydrate and carbolic acid equal parts, liquefied with alcohol, is a powerful obtundent of hypersensitive dentin. It is an irritant caustic (Robinson's Remedy).

Potassium iodid in ointment is a useful application to indurations about the jaws, left as the result of chronic inflammations.

℞. Potassii iodid.,	gr. xx ;
Cerat. simp.,	ʒj.—M. et ft. unguent.

Potassium iodid is administered internally in cases of mercurial stomatitis, pericementitis, and periostitis, as an eliminative agent.

Potassium permanganate is an effective oxidizing deodorant and antiseptic ; its germicidal power is very doubtful. In contact with organic matter it gives up its oxygen and is reduced to manganese oxid. It is a chemical antidote of morphin, and of snake-venom.[1] It is used in claret-colored solutions to deodorize an offensive mouth.

Pyrozone. (See Hydrogen Dioxid.)

Quercus.

A tincture of quercus alba, or white-oak bark, diluted, is used as an astringent to reduce the tumidity of the gums.

Quillaia, Soap-bark,

is used powdered, as a substitute for powdered Castile soap in dentifrices.

*Resorcin, $C_6H_4(HO)_2$.

A close analogue of carbolic acid, C_6H_5HO. Is more poisonous than carbolic acid, and does not act so promptly.[2] Its uses are similar to those of carbolic acid. It is more soluble in water than the latter.

Saccharin, $C_6H_4(CO)(SO_2)NH$, Benzoyl-sulphonic Imide.

A coal-tar derivative more than two hundred times sweeter than

[1] S. Weir Mitchell. [2] Brunton.

sugar; it is antiseptic, checking fermentations. For this reason a minute portion is used as the sweetening agent in mouth-washes, instead of sugar. The solution must be made with alcohol.

Salol, Phenyl Salicylate, $C_6H_4(OH)(CO.OC_6H_5)$.

A white crystalline powder insoluble in water, melting at about 105° F.; it remains fluid for some time after reduction to a temperature below that of the body. It is decomposed by alkalies into carbolic and salicylic acids. It has been used in its melted state as a root-canal filling. Administered internally in 5 gr. doses to relieve rheumatic pains about the jaws.

Silver.

Silver nitrate (argenti nitras) is employed in all strengths from a 1 per cent. solution to the fused nitrate—the solid stick or lunar caustic. Nitrate of silver exposed to sunlight undergoes decomposition and is reduced to the oxid. Brought in contact with albuminous substances, silver nitrate combines with them, forming the albuminate of silver. It has been held by writers upon pharmacology that the action of silver nitrate applied to a surface was very superficial; that a film of silver albuminate formed and the chemical reaction ceased. Experiments by Truman[1] have shown that its penetrative power is very great as compared with other coagulants. Placed at one end of a capillary tube containing a solution of albumin, silver nitrate quickly brings about coagulation to the extremity of the tube.

It has long been used as an empirical remedy for traumatic erysipelas, for which it has been held to be a specific. To act as a germicide in such cases its action must be penetrating, as the streptococci of erysipelas are deeply situated.

Nitrate of silver, in saturated solution, is used to arrest caries in deciduous teeth (see Chapter XXVII.) and also to destroy hypersensitivity of dentin; its deeply penetrative action suggests caution in this direction. It is never used except in posterior teeth because of its staining, owing to the reduction of the silver albuminate to silver oxid.

In 5–10 per cent. solution it is used as a wash for pyorrhœa pockets in which pus-formation and congestion are persistent.

Other salts of silver, notably the **citrate** and **lactate**, have been shown to be prompt and effective germicides. The silver citrate is soluble in the proportion of about 1 : 3800 in water, in which strength it is sufficiently active to sterilize the cavities of abscesses and as a wash in inflammatory and suppurative affections about the jaws. The stains upon the enamel are easily removable by means of common abrasives.

[1] *Proc. Academy of Stomatology*, 1895.

The powdered silver citrate should make a valuable addition to the melted paraffin for use as a canal-filling in posterior teeth.

The lactate of silver, soluble 1 : 15 in water, is slightly irritating.

Sodium.

The sodium salts are usually regarded as having therapeutic properties similar to those of the potassium salts. Bartholow[1] maintains strongly that they differ in essential properties; the sodium salts are more diffusible, are less irritating to mucous membranes, and are less toxic to the tissues, including the cerebral and circulatory centres. Potassium salts are more active in promoting destructive metamorphosis, and in removing inflammatory growths and promoting excretion. What has been said of the dental uses of potassium bromid, carbonate, and hydrate, and metallic potassium, applies, however, to the corresponding salts of sodium.

Sodium biborate, borax, $Na_2B_4O_7 + 10H_2O$, is used in solution with glycerin in the treatment of catarrhal and ulcerative stomatitis of children.

Sodium bicarbonate, $NaHCO_3$, is used in carious cavities to lessen dentinal hypersensitivity by neutralizing the acids to which the condition is probably due.[2]

Sodium phenate, phenol sodique, C_6H_5NaO, is used as an antiseptic and styptic. It is a local anæsthetic; its common uses are those of carbolic acid, but it is without the cauterant action of the latter.

Sodium peroxid, Na_2O_2, in contact with organic matter sets free nascent oxygen; the sodium oxid left is quickly converted into sodium hydrate, having the properties of that substance. Solutions must be made gradually and in ice-cold *distilled* water, as the heat of combination raises the temperature of the water, and oxygen escapes. A saturated solution is first made, which is afterward diluted with distilled water. Powdered sodium dioxid is hygroscopic and is rapidly decomposed upon exposure to the air. In saturated solution (applied by means of aluminum, gold, or platinum points) it is used in the treatment of putrescent pulps, and as a bleaching agent for discolored dentin. It possesses the properties of sodium hydroxid and nascent oxygen.

Sodium sulfite, $Na_2SO_3 + 7H_2O$. A mixture of sodium sulfite, 10 parts, and boric acid, 7 parts, made dry, is inserted into the cavity of a tooth in which the dentin is to be bleached; when water is applied a reaction occurs, sulfurous acid being liberated:

$$2H_3BO_3 + 3Na_2SO_3 = 2Na_3BO_3 + 3H_2O + 3SO_2.$$[3]

[1] International Clinics, 1898, vol. iv., 7th series. [2] Truman,

[3] Kirk, *American Text-book of Operative Dentistry.*

Unlike sodium dioxid and chlorin, this substance SO_2, bleaches by reduction, abstracting the oxygen from the pigment-molecule.

Sodium silico-fluorid (salufer), Na_2SiF_6. Sodium silicate, liquid silex, Na_2SiO_3. The latter substance has antiseptic properties. In the former the antiseptic and deodorant properties are marked. Sodium silico-fluorid is soluble in about the proportion of 1 per cent. in water. In this strength it has been used in the treatment of putrescent pulps.

Sozoiodol, $C_6H_2(HSO_3)I_2OH$,

is a compound of iodin with paraphenol sulphonic acid. It is used as an antiseptic in pyorrhœa pockets. Miller's experiments[1] show that sozoiodol salts are but weak dental antiseptics.

Thymol,

a stearoptene derived from the volatile oil of thyme, is methy-propyl-phenol, $C_6H_3(CH_3)(C_3H_7)HO$. It is soluble in alcohol and ether. It possesses the properties of a methyl phényl—*i. e.*, is markedly antiseptic and analgesic. It is extensively used as an analgesic and antiseptic in the treatment of diseases of the pulp.

Trichlorphenol, $C_6H_2Cl_3HO$,

is a powerful and penetrating antiseptic, stronger than carbolic acid. "It possesses the power of penetrating pulp-tissue rapidly, thoroughly hardening it and imparting to it a pink to red color. Its pulp-preserving power is very high."[2] It is applied to pulps which have been but partially devitalized by arsenic, to sterilize them and complete their devitalization.

Dental Varnishes.

℞. Gum sandarac, ℥ij ;
Alcohol, Oj.
Mix, and aid solution with heat. Filter solution through cotton-wool.

This is used in the dental laboratory as a separating medium and to varnish casts.

Pellets of cotton-wool dipped in the sandarac solution are used to cover medicinal applications in teeth and to prevent the ingress of foreign materials. Being in no degree antiseptic, and becoming foul after twenty-four hours or longer, soft gutta-percha preparations (temporary stopping) have largely superseded cotton and sandarac.

[1] *Dental Cosmos*, 1890. [2] Miller, *Dental Cosmos*, 1895.

Renewed every day, cotton and sandarac dressings are useful to press away the gum overhanging the margins of cavities, to check alveolar hemorrhage, and to temporarily close the crowns of teeth under treatment. It is also used to varnish the walls of prepared cavities prior to inserting plastic fillings, to prevent the irritation incident to the application of zinc oxychlorid cement, and to prevent the irritation of zinc phosphate in a soft state, due to its acid reaction in this condition; and also to prevent the action of the acid upon dentinal walls. It is also used to prevent the action of acid substances which may be present as impurities in zinc-cement fluids, notably, the acid sodium phosphate, dihydrogen sodium phosphate (H_2NaPO_4).

SHELLAC VARNISH.

The coloring, separating medium of the dental laboratory.

℞. Gum shellac,	℥ij;
Alcohol,	Oj.—M.
Aid solution by heat.	

In its unmodified form it has but little use aside from that of the first or coloring varnish applied to plaster impressions. It is used to apply to enamel-surfaces which have been cleansed with chloroform, to secure greater adhesion between orthodontic rings and the teeth. The tooth to which a "regulating ring" is to be applied is washed with chloroform, dried, and a coating of shellac varnish given to the part to be covered by the ring. The latter is painted with zinc-phosphate cement, which is also applied over the shellac coating, and the ring pressed into place; the parts should be kept dry until the cement is hard. Combined with appropriate antiseptics shellac varnish makes an admirable protective covering to abraded surfaces of the mouth which might invite infection.

The following formula (steresol) has been given by M. Berlioz,[1] of Grenoble, France, for an adhesive, anæsthetic, antiseptic, impermeable coating to be applied over abrasions, wounds, etc., of the tissues of the mouth.

℞. Purified gum lac,	270 gms., about	℥ ix;
Purified gum benzoin,	10 " "	℥$\frac{1}{3}$;
Balsam of tolu,	10 " "	℥$\frac{1}{3}$;
Oil of cinnamon (Chinese),	6 " "	℥$\frac{1}{5}$;
Acid. carbolic.,	100 " "	℥iij;
Saccharin,	6 " "	℥$\frac{1}{5}$;
Alcohol,	q. s. ft. one liter, "	Oij.

[1] *Dental Cosmos*, 1895.

Veratrum Viride.

Its tincture, in from 2 to 4 drop doses, is used to reduce the force of the circulation in the early stages of sthenic inflammations; it is to be preferred to aconite in this connection, because it causes vomiting before a lethal dose has accumulated in the system.[1]

Veratrina.

The paralyzant alkaloids of veratrina sabadilla, not of veratrum viride. There are at least three alkaloids entering into the composition of commercial veratrina (U. S. Dispensatory). The U. S. P. (p. 204) ointment is used to rub over the temporo-maxillary articulation and over the masseter muscle to relieve spasm due to difficult eruption of the lower third molar. It should be used in small amount and be kept away from the mouth and eyes, as it is actively poisonous.

Zinc.

Zinc derivatives are among the most important therapeutic agents of dentistry.

Zinc chlorid, $ZnCl_2$, is very deliquescent; it abstracts moisture from the atmosphere and becomes fluid soon after exposure. It combines readily and actively with albuminous substances, forming zinc albuminate. Applied to living tissues, the combination occurs promptly and is attended with much pain.

Truman found[2] that its penetrative power in saturated solution was greater than that of any other coagulant. In from 10 to 20 per cent. solution it is a powerful astringent. The degree of astringency may be graded by varying the percentage-strength of the solution. In 1 to 5 per cent. solution this substance is a stimulant, astringent, and germicide. It is used in full strength to obtund the hypersensitivity of peripheral dentin. In saturated solution it is the fluid of zinc-oxy-chlorid cement:

$$ZnO + ZnCl_2 + H_2O = 2ZnClHO.$$

A paste which hardens and maintains an antiseptic action for some time after hardening. Zinc chlorid, in 50 per cent. solution, or stronger, is used to coagulate the contents of the dentinal tubuli after devitalization and removal of the dental pulp. As a germicide, astringent, and stimulant, it is a useful agent in the treatment of pyorrhœa alveolaris; also in cases of chronic abscess with serous exudations.

Zinc iodid, in 20 per cent. solution, is an excellent application to pyorrhœa pockets in cases of phagedenic pericementitis, after the

[1] Hare.

[2] *Proc. Academy of Stomatology*, 1895.

removal of calculi and the washing of the pockets (Harlan). It is an excellent stimulant wash for the same, in 2 per cent. solution for subsequent applications.

Zinc oxid is the basal powder of the zinc cements, the oxychlorid, the phosphate, and zinc oxysulfate. It may contain arsenic trioxid as an impurity, in which event cements made of it may kill the dental pulp. An ointment of zinc oxid is used upon abrasions about the lips.

The **orthophosphate of zinc**, a combination of orthophosphoric acid with zinc oxid, is the most important of dental cements:

$$3ZnO + 2H_3PO_4 = Zn_3(PO_4)_2 + 3H_2O.$$

Zinc sulfate in saturated solution, if combined with a powder of zinc oxid, forms an oxysulfate of zinc, a body having about the hardness and porosity of plaster of Paris; this compound is used to protect fully or partially exposed pulps. Zinc sulfate in 10 per cent. solution is a useful astringent wash in stomatitis.

BLACK'S 1–2–3 MIXTURE.

Oil of cinnamon,	1 part;
Carbolic acid,	2 parts;
Oil of gaultheria,	3 parts.

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